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Finerenone Use in Heart Failure Patients with Type 2 Diabetes and Kidney Disease: An Analysis of the FINE-TURK Registry

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Abstract

Background: Finerenone, a non-steroidal mineralocorticoid receptor antagonist, improves cardiorenal outcomes in patients with type 2 diabetes mellitus and chronic kidney disease. However, real-world data on its use in patients with concomitant heart failure remain limited. This study aimed to evaluate the baseline characteristics and short-term renal safety and efficacy of finerenone in patients with type 2 diabetes, kidney disease, and heart failure from the FINE-TURK Registry.

Methods: This was a retrospective, multicenter, observational registry analysis conducted across 56 centers in Türkiye. Among 1,168 patients initially enrolled, 918 patients with available heart failure status and ejection fraction data were included. Patients were categorized according to the presence of heart failure, and heart failure patients were further classified as HFrEF or HFmrEF/HFpEF. Three-month changes in serum potassium, estimated glomerular filtration rate, and albuminuria were assessed. Safety endpoints included serum potassium ≥5.5 mEq/L and eGFR ≤25 mL/min/1.73 m² at three months. The primary renal efficacy endpoint was a >30% reduction in albuminuria from baseline.

Results: Of 918 patients, 133 had heart failure. Patients with heart failure were older and had lower baseline eGFR compared with those without heart failure. They were also more likely to receive a lower starting dose of finerenone. At three months, changes in serum potassium, eGFR, and albuminuria were not significantly different between patients with and without heart failure. No excess risk of hyperkalemia or worsening renal function was observed in the heart failure group. In adjusted analyses, heart failure was associated with a greater likelihood of achieving a >30% reduction in albuminuria. No significant differences in short-term renal safety or efficacy outcomes were observed between HFrEF and HFmrEF/HFpEF subgroups.

Conclusions: In this nationwide real-world registry, patients with heart failure who were treated with finerenone had a higher-risk baseline profile, including older age and lower eGFR. Despite this, finerenone was not associated with increased short-term renal safety concerns and was linked to a clinically meaningful reduction in albuminuria. These findings support the short-term renal safety and potential efficacy of finerenone in patients with type 2 diabetes, kidney disease, and heart failure.

Introduction

Heart failure affects an estimated 64 million people globally and is one of the leading causes of death from cardiovascular causes (1,2). In patients with heart failure, sustained activation of renin-angiotensin-aldosterone system is associated with salt and water retention, increased afterload and progressive remodeling that furthers the initial damage and leads to disease progression and death (3). Aldosterone is a steroid hormone that activates mineralocorticoid receptors present in renal and cardiac tissue and promotes inflammation and fibrosis in both organs (4). Although activation of this system is most prominent in patients with reduced ejection fraction (EF), some degree of activation is also present in patients with heart failure (HF) and relatively preserved left ventricular contractility (5).

Studies conducted in late 1990's and early 2000's have unequivocally demonstrated the usefulness of steroidal mineralocorticoid blockers (MRAs), namely spironolactone and eplerenone, for treatment of patients with HF and an EF below 40% (6,7). Although small-scale studies have also suggested a benefit with steroidal MRAs in patients with minimally reduced or preserved EF, the landmark Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist (TOPCAT) trial failed to demonstrate the usefulness of spironolactone in this patient group (8-10). Although the explanation for this latter result focused heavily on geographical variations of the trial cohort, a simpler explanation would be the inadequate receptor affinity and clinically relevant side effects of spironolactone which could have offset the benefits of mineralocorticoid antagonism in patients with preserved EF (11,12).

Finerenone is a new generation non-steroidal MRA with increased receptor affinity and fewer significant side effects that limits the usefulness of steroidal MRAs (13). The usefulness of finerenone on both kidney and cardiovascular outcomes, including a reduction in worsening kidney function and a reduction in HF hospitalizations were demonstrated in the combined analysis of two landmark trials, namely Cardiovascular Events with Finerenone in Kidney Disease and Type 2 Diabetes (FIGARO-DKD) and Effect of Finerenone on Chronic Kidney Disease Outcomes in Type 2 Diabetes (FIDELIO-DKD) (14-16). More recently, finerenone was shown in the Finerenone in Heart Failure with Mildly Reduced or Preserved Ejection Fraction (FINEARTS-HF) as the second treatment that could effectively reduce a composite of mortality and HF-related adverse outcomes (17). While data from the large randomized-controlled trials established finerenone as an important component of the treatment of patients with HF, there is a paucity of real-world data that could support these findings.

Finerenone in Turkish patients (FINE-TURK) is a multi center registry study that aimed to collect real-life data from patients with type 2 diabetes (T2DM) and evidence for nephropathy in whom finerenone was initiated for renal and cardiovascular protection. Present study reports the baseline characteristics of HF patients included in the registry and analyzed similarities and differences of HF cohort with the overall study population. As secondary aims, we sought to analyze short-term (3 months) outcomes on safety and efficiency of finerenone for renal protection in the HF cohort and whether there were any differences between HF and non-HF patients regarding to these outcomes.

Methods

This registry was a retrospective observational study that collected data from patients in whom finerenone was indicated for cardiovascular and renal protection, based on previous trial data. The registry was performed in 56 centers across all the geographical regions in Turkey. Data collection was initiated on January 2025 and and last follow-up visits will be conducted on June 2025. Inclusion criteria were being over 18 years old, a prior diagnosis of T2DM and presence of at least one evidence for nephropathy: i) estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73 m^2 or ii) albuminuria defined as presence of urinary albumin >30 mg/daily or >30 mg albumin/mg creatinine in a spot urinary sample on at least two samples. Patients with an eGFR ≤25 ml/min/1.73 m^2 or undergoing renal replacement therapy for any indication, patients with a potassium concentration over ≥5.5 mEq/L and

patients with previously known nephropathy with a non-diabetic etiology were excluded. In addition to patients who were deemed suitable for finerenone treatment based on initial clinical assessment, patients who were receiving finerenone for the last 3 months were considered eligible for inclusion if: i) the patient was seen by a given investigator at a study center prior to receiving finerenone and finerenone was initiated by the same investigator based on criteria compatible with enrollment criteria, and ii) data for baseline variables were present at the time of study commencement. For this present analysis, no additional inclusion / exclusion criteria were used other than missing data for the presence and phenotype of HF. Patients with HF were included to the present study regardless of their EF, and the analysis were done including patients with reduced ejection fraction.

This study was conducted according to the 1975 Declaration of Helsinki and its subsequent revisions, and all patients included to the present analysis provided their written informed consent prior to enrollment. The study was approved by a scientific research ethics committee (Decision No: 2024/010.99/11/23).

Study Protocol

This was a retrospective, multicenter, observational real-world registry analysis of patients in whom finerenone had been prescribed for cardiovascular and renal protection on the basis of previous trial evidence and routine clinical judgment The. registry was conducted in 56 centers across all geographic regions of Türkiye. For the present analysis, baseline and three-month data were retrospectively abstracted from institutional medical records and entered into the registry database.

The inclusion criteria were age >18 years, a prior diagnosis of T2DM, and the presence of at least one indicator of nephropathy. Albuminuria, defined as urinary albumin-to-creatinine ratio >30 mg/g in a spot urine sample on at least two occasions. The exclusion criteria were eGFR ≤25 mL/min/1.73 m², renal replacement therapy for any indication, serum potassium ≥5.5 mEq/L, and previously known nephropathy of non-diabetic etiology.

Patients who had received finerenone in routine clinical practice were eligible if: (i) they had been evaluated at a participating study center before finerenone initiation; (ii) finerenone had been started according to criteria compatible with the registry eligibility criteria; and (iii) baseline and relevant follow-up data were available in the medical records. For the present analysis, no additional inclusion or exclusion criteria were applied other than the availability of data regarding HF status and HF phenotype. Patients with HF were included irrespective of EF, including those with reduced EF.

The study was conducted in accordance with the 1975 Declaration of Helsinki and its subsequent revisions. The study protocol, including retrospective data collection procedures, was approved by the relevant scientific research ethics committee (Decision No: 2024/010.99/11/23). Patient consent procedures were conducted in accordance with the ethics committee requirements and applicable local regulations. Patients in whom recent data on EF was lacking were excluded from the present analysis. Details of study design and workflow can be found in Supplementary Figure 1.

Patient follow-up and outcomes

Patients were recalled at 1st, 3rd, 6th and 12th months for follow-ups and laboratory analyses including blood chemistry and urine analysis for microalbuminuria were repeated. In case patients could not be contacted, patients 'relatives were contacted with telephone to collect clinical outcomes data. For this present analysis, primary safety endpoints were potassium ≥5.5 mEq/L or eGFR ≤25 ml/min/1.73 m^2 at 3rd month, and a drop of >30% in urinary albumin concentration relative to baseline were considered as primary efficiency endpoints.

Statistical Analysis

Continuous variables were given as mean ± SD or as median [IQR] depending on the pattern of distribution, while categorical variables were given as percentages. Patterns of distribution for continuous variables were analyzed using Shapiro-Wilk test, visual inspection of histograms and Q-Q plots. Student's T or Mann-Whitney U tests were used as appropriate for statistical analysis of continuous data, while chi-squared or Fisher's exact tests were used for categorical data. For continuous variables with more than one measurement obtained at different time points, paired-samples T test or Wilcoxon signed rank tests were used as appropriate. Effect sizes for paired analyses were assessed using Cohen's d or with rank biserial correlation. To understand whether changes in renal safety and efficacy parameters were different between HF and non-HF groups at third month, separate ANCOVA models were constructed with using baseline values as covariates. Univariate and multivariate logistic regression models were built to understand whether changes in safety and renal efficacy outcomes differed between the two groups at 3rd month. For the multivariable models; age, gender and body mass index were used as adjustment covariates. Crude and adjusted hazard ratios and confidence intervals were calculated and first plots were constructed to summarize results.

For subgroup analyses, patients with HF were categorized into two subgroups (EF ≤40% and EF >40%) based on ejection fraction. Same statistical tests summarized above were used for subgroup analyses. For all statistical analyses, a p value <0.05 were accepted as statistically significant. All statistical analyses were done using Jamovi statistical software (The jamovi project (2025). jamovi. Version 2.7 for MacOS. Retrieved from https://www.jamovi.org).

Results

Out of 1,168 patients initially included to the initial cohort, 77 cases were excluded due to insufficient data and a further 173 patients were excluded due to uncertainty on the presence of HF and/or EF. As such, the final analysis were done using 918 patients. Of those 918 patients, 133 patients (14.5%) had HF, with 89 of these 133 patients (66.9%) having a preserved or minimally reduced EF (HFpEF/HFmrEF) and a further 44 patients (33.1%) having HF with reduced EF (HFrEF).

Table 1 summarizes demographic, clinical and labaratory data at baseline for patients with or without HF. On overall, patients with HF were older, more likely to have concomitant coronary artery disease with

or without previous revascularization and lower eGFR as compared to those without HF. Although there was a trend towards lower urinary albumin concentration in HF patients, this finding did not reach statistical significance.

Baseline data on medications and finerenone dosing were summarized in Table 2. ACE inhibitors, angiotensin receptor inhibitors (ARBs) and SGLT2 inhibitors were ubiquitously used in both groups, with no differences between HF and non-HF groups. As expected, HF patients were more likely to be on beta-blockers and furosemide than patients without HF. Roughly two-thirds of patients within both groups were on a maximum dose of a given renin-angiotensin system (RAS) inhibitor, while median duration of RAS inhibitor use was longer in HF patients. Compared to patients without HF, HF patients were more likely to receive a lower dose of finerenone at enrollment.

Changes in potassium, eGFR and albuminuria 3 months after finerenone were given on Table 3 and Figure 1. Serum potassium has increased and both eGFR and albuminuria have decreased in both groups, and the changes were independent of the presence or absence of HF for all parameters analyzed. Notably, the decline in eGFR was not statistically significant in the HF group.

Figure 2 summarizes crude and adjusted hazard ratios for primary safety and renal efficiency endpoints. After adjustment for age, gender and body mass index; a drop in urinary albumin at 3rd month was more common in patients with HF as compared to those without, although the there were no differences in the number of patients achieved a microalbumin concentration <300 mg/day in both groups. There were no differences in safety endpoints between the two groups in unadjusted or adjusted analyses.

Subgroup analyses

Baseline data for patients within the HFrEF and HFpEF/HFmrEF groups were summarized in Supplementary Tables 1 and 2. HFrEF patients tend to have lover systolic blood pressure, were more likely to be on furosemide and were less likely to be on a maximal dose of a given RAS inhibitor than patients with HFpEF/HFmrEF. Other baseline characteristics, including eGFR, were similar across the two groups.

Changes in serum potassium, eGFR and albuminuria in HF subgroups were given in Supplementary Table 3. In both groups, serum potassium concentrations were increased and albuminuria were reduced at third month, and no interactions were seen between the observed changes with the HF subgroup. Notably, there were no statistically significant changes in eGFR in either subgroup, although there was a numerical decrease in both HF subgroups.

Discussion

In this preliminary report of a nationwide registry, we have assessed the characteristics of HF patients with T2DM in whom finerenone was added to the treatment regimen for prevention of adverse renal and cardiovascular outcomes. Present findings indicate that HF patients are older and have worse renal

function at baseline, but despite that presence of HF does not confer any additional risk for hyperkalemia or worsening renal function during a 3-months follow-up. In contrast, there was a significant 64% increase in the HF group for a clinically relevant reduction in albuminuria, suggesting a possible additional benefit on renal outcomes in this patient population. A summary of the main findings can be found in the Graphical Abstract.

Development of chronic kidney disease is a harbinger of significant morbidity and mortality in T2DM patients, and the landmark randomized trials of finerenone, namely FIDELIO-DKD and FIGARO-DKD, were designed to test the hypothesis that finerenone would be effective for preventing the development of nephropathy in these patients (14-16, 18). 7.8% (571 patients, with 290 patients assigned to the finereone arm) of the patient population included in the FIGARO-DKD had HF at baseline (19). Although most of the baseline characteristics of HF patients presented in this report matches closely to that of FIGARO-DKD, two important distinctions were present: i) FIGARO-DKD cohort did not include patients with a reduced EF, while roughly one-third of HF patients included in the FINE-TURK cohort were composed of HFrEF patients, and ii) HF patients in FINE-TURK study had a worse kidney function than that of the FIGARO-DKD cohort (eGFR 48.0 ±17.8 vs. 63.4 ± 21.7) (19). Although finerenone is currently not indicated in patients with HFrEF; given that finerenone is a theoretically better MRA with proven renoprotective effects and is effective for reducing adverse events in patients with an EF over 40%, it is reasonable to consider that finerenone is likely to be used in real-world clinical settings where steroidal MRAs are either contraindicated or not tolerated. Indeed, this assumption is currently tested in the FINALITY-HF trial (NCT06033950), which is expected to end in 2028. Although the overall sample size of the HFrEF cohort was relatively small (44 patients), FINE-TURK registry will nonetheless provide an earlier glimpse on the use of finerenone in HFrEF patients when final results are available.

A major concern for the use of MRAs is the increased risk of hyperkalemia and an early reduction in eGFR, which mandates close follow-up after initiating these drugs (20). The molecule structure of finerenone differs from the steroidal MRAs spironolactone and eplerenone, thus allowing a better receptor selectivity and less side effects (13). However, pooled analysis of the FIGARO-DKD and FIDELIO-DKD trials indicate that clinically relevant hyperkalemia (12.0%) as well as hyperkalemia leading to permanent stopping of the medication (1.7%) was more common in the finerenone arm. Present finding suggest that although treatment-related increases in potassium was seen in both HF and non-HF patients, there were no differences between the groups in terms of absolute increases in potassium or the frequency of cases with clinically-relevant (≥5.5 mEq/L) hyperkalemia. Indeed, the frequency of significant hyperkalemia was numerically lower in the HF arm, and a similar finding was noted in a subgroup analysis of the FIGARO-DKD study (19). To note, present analysis included patients with HFrEF and HF patients had an overall lower eGFR than non-HF patients, which is reassuring given than these patients would be expected to have a higher incidence of hyperkalemia following MRAs.

Present results suggest a numerical decrease in eGFR three months after the initiation of finerenone, but this finding was not statistically significant and there were no interactions between the presence of HF and the drop in GFR at three months. A small decrease in eGFR should be anticipated after finerenone

and this drop does not translate into worse renal or cardiovascular outcomes, as demonstrated in a prespecified analysis of the FINEARTS-HF trial (21). Such transient alterations are common after initiation of renoprotective drugs, such as ACE inhibitors or sodium-glucose cotransporter-1 inhibitors, and this finding is not an indication for long-term worsening of renal function (22,23). Present findings should be considered as reassuring from a safety perspective given that the initial change in GFR was neither statistically nor clinically relevant in this real-world data.

In contrast, we have found a statistically significant reduction in albuminuria in both groups, and the odds for a clinically relevant drop was statistically higher in the HF group as compared to non-HF patients. To the best of our knowledge, this is the first finding suggesting that finerenone might have a selectively positive impact on renal functions in HF patients in a patient cohort at high risk for adverse renal events (i.e. with T2DM and nephropathy). A recent prespecified analysis of the FINEARTS trial found that although finerenone was associated with a significant 30% drop in albuminuria compared to placebo in HF patients, the treatment was not related to a modification of adverse renal events (24). As the FINEARTS-HF cohort included patients at a relatively low risk of worsening renal failure, this latter results should not be generalized to those at a higher risk for renal events. To note, present results only show a reduction in a surrogate marker of adverse renal events over a relatively short period of time in a nonrandomized registry cohort and therefore considered only as hypothesis-generating. Nonetheless, this encouraging data could be considered as a basis for further studies and trials investigating this possibly selective effect of finerenone on HF patients at high risk for renal events.

Neurohormonal blockade remains as the cornerstone of the treatment of HF, particularly HFrEF, and reaching the maximum tolerated dose for RAS inhibitors, including MRAs, is required to improve event-free survival (25). Although both doses of finerenone were found to be effective for nephroprotection, preliminary data from the Phase IIb MinerAlocorticoid Receptor antagonist Tolerability Study-Heart Failure (ARTS-HF) study suggested an overall decrease of adverse events in the 20 mg, thus emphasizing the need for reaching the maximum dose of finerenone in these patients (26). Despite that, present real-world findings suggest that the dose of finerenone was more likely to be sub-maximal in the HF cohort, thus potentially limiting the usefulness of these drugs for preventing major cardiorenal events. One possible reason for this finding is that in contrast to the above-mentioned analysis of the FIGARO-DKD trial, HF patients in this present cohort had a lower mean eGFR value at baseline, which might have sparked an anxiety in caregivers for further drops in eGFR that limited the dose of both RAS inhibitors and finerenone (19). However, there is no evidence to suggest that RAS inhibitors were associated with worse renal outcomes even in patients with “advanced” renal disease, and available evidence indeed suggests continuing benefit with finerenone in the subgroup of patients with eGFR <60 ml/min/1.73 m^2 and significant albuminuria (27,28). Thus, this “anxiety” for further renal decline is not evidence based and may harm patients as sub-maximal dosing prevents fully benefiting from these drugs and may increase untoward cardiorenal outcomes. To note, present findings only reflect baseline dosing for finerenone and changes in dosing during the registry period will be reported when results are available.

Present analysis have several strengths and limitations. The sample population was enrolled across all geographic locations across the country, thus increasing the generalizability of the results. Some patients were already on finerenone at the first outpatient visit and were enrolled retrospectively at the first or third month to increase the sample size. However, care has been taken to ensure that patients would be eligible for inclusion based on the prior clinical and laboratory findings and data at baseline was sufficient for inclusion. While this was a nationwide registry, sample size (and particularly the sample size for patients with HF) was relatively limited, owing to a lack of reimbursement of finerenone by the social security system. At the time of writing this manuscript, adequate data were only available for on short-term (3rd month) outcomes on safety outcomes and albuminuria. As such, data on hard end points will be reported in a separate paper as more results become available.

Conclusion

In this baseline analysis of the real-world data on the use of finerenone in T2DM patients at high risk for adverse renal events, we found that patients with HF had a better reduction in albuminuria as compared to non-HF patients, despite having a lower eGFR at baseline. Also, available safety data at 3rd month does not indicate an additional risk in HF patients for hyperkalemia or a reduction in glomerular filtration with finerenone. Although present analysis only reported short-term data on the use of finerenone in HF patients with T2DM and at high risk for adverse renal events, available data nonetheless confirms the safety and possible efficacy of finerenone in such patients.

Declarations

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Tables

Table 1. Baseline demographic and clinical characteristics of patients with and without heart failure. HF: Heart failure, BP: Blood pressure, CVA: Cerebrovascular accident, GFR: Glomerular filtration rate.

Characteristic	Patients without HF (n=785)	Patients with HF (n=133)	P value
Age (y)	59.5 ± 11.5	66.3 ± 10.3	<0.001
Gender (%Female)	45.4%	42.1%	0.487
Body mass index (kg/m^2)	31.6 ± 14.5	29.6 ± 6.1	0.079
Systolic BP (mmHg)	134.0 ± 15.5	133.0 ± 17.0	0.416
Diastolic BP (mmHg)	81.0 ± 9.4	78.4 ± 10.6	0.015
Smoking			0.035
• Not used / ex user (%)	515 (70.8%)	95 (80.5%)	
• Active smoker (%)	212 (29.2%)	23 (19.5%)	
Physical activity			0.004
• None / Infrequent (%)	558 (80.4%)	95 (92.3%)	
• Regular (%)	136 (19.6%)	8 (7.7%)	
Hypertension (%)	730 (93.0%)	128 (96.2%)	0.161
Previous CVA (%)	56 (7.1%)	18 (13.7%)	0.010
Coronary artery disease (%)	205 (26.1%)	108 (82.4%)	<0.001
Previous revascularization (%)	100 (12.8%)	87 (66.4%)	<0.001
Peripheral artery disease (%)	52 (6.7%)	20 (15.7%)	<0.001
Estimated GFR (ml/min/1.73 m2)	60.4 ± 24.7	48.0 ± 17.8	<0.001
Hemoglobin (g/dl)	13.4 ± 1.9	13.0 ± 2.1	0.013
Sodium (mEq/L)	139.0 ± 2.7	139.0 ± 3.4	0.133
Potassium (mEq/L)	4.5 ± 0.4	4.4 ± 0.5	0.008
Hemoglobin A1c (%)	7.3 [6.5 - 8.5]	7.9 [6.8 - 9.1]	0.006
Albuminuria (mg/d)	722.0 [285.0 - 1600.0]	477.0 [200.0 - 1394.0]	0.053

Table 2. Baseline medical therapy of patients with and without heart failure. HF: Heart failıre, RAS: Renin-angiotensin system, SGLT2: Sodium-glucose linked transporter.

Characteristic	Patients without HF (n=785)	Patients with HF (n=133)	P value
Medications			
• ACE inhibitors (%)	369 (47.4%)	53 (39.8%)	0.105
• ARBs (%)	357 (45.8%)	65 (49.2%)	0.459
• Calcium CBs (%)	370 (47.4%)	55 (41.7%)	0.224
• Beta blockers (%)	285 (36.5%)	104 (79.4%)	<0.001
• Furosemide (%)	82 (10.5%)	66 (50.4%)	<0.001
• SGLT2 inhibitors (%)	683 (87.1%)	117 (88.0%)	0.785
• GLP1 agonists (%)	41 (5.2%)	5 (3.8%)	0.666
RAS inhibitor dose			0.467
• Submaximal (%)	266 (36.2%)	38 (32.8%)	
• Maximal (%)	468 (63.8%)	78 (67.2%)	
RAS inhibitor duration (mo)	96.0 [36.0 - 144.0]	120.0 [60.0 - 180.0]	0.004
SGLT2 inhibitor duration (mo)	15.0 [8.0 - 30.0]	15.0 [8.0 - 31.8]	1.000
Finerenone starting dose			0.026
• 10 mg	655 (83.5%)	121 (91.0%)	
• 20 mg	130 (16.5%)	12 (9.0%)	

Table 3. Changes in serum potassium, estimated glomerular filtration rate and albuminuria at 3rd month in patients with and without HF. ES: P_{change}: P value for change in paired variables, Effect size, P_{int}: P value for interaction, HF: Heart failure.

Characteristic	At baseline	3-months follow up	P_{change}	ES	P_{int}
Serum potassium (mEq/L)					
No HF (n=490)	4.48 ± 0.37	4.79 ± 0.45	<0.001	-0.677	0.668
HF (n=79)	4.40 ± 0.44	4.73 ± 4.80	<0.001	-0.671	
Estimated glomerular filtration rate (ml/min/1.73 m2)					
No HF (n=436)	60.2 ± 23.7	58.0 ± 25.5	<0.001	0.177	0.695
HF (n=69)	49.5 ± 15.6	47.4 ± 15.7	0.094	0.218	
Albuminuria (mg/d)					
No HF (n=383)	790.0 [330.0 - 1600.0]	450.0 [151.0 - 1042]	<0.001	0.758	0.255
HF (n=42)	552.0 [367.0 -1395.0]	465.0 [156.0 - 826.0]	<0.001	0.801	

Figures

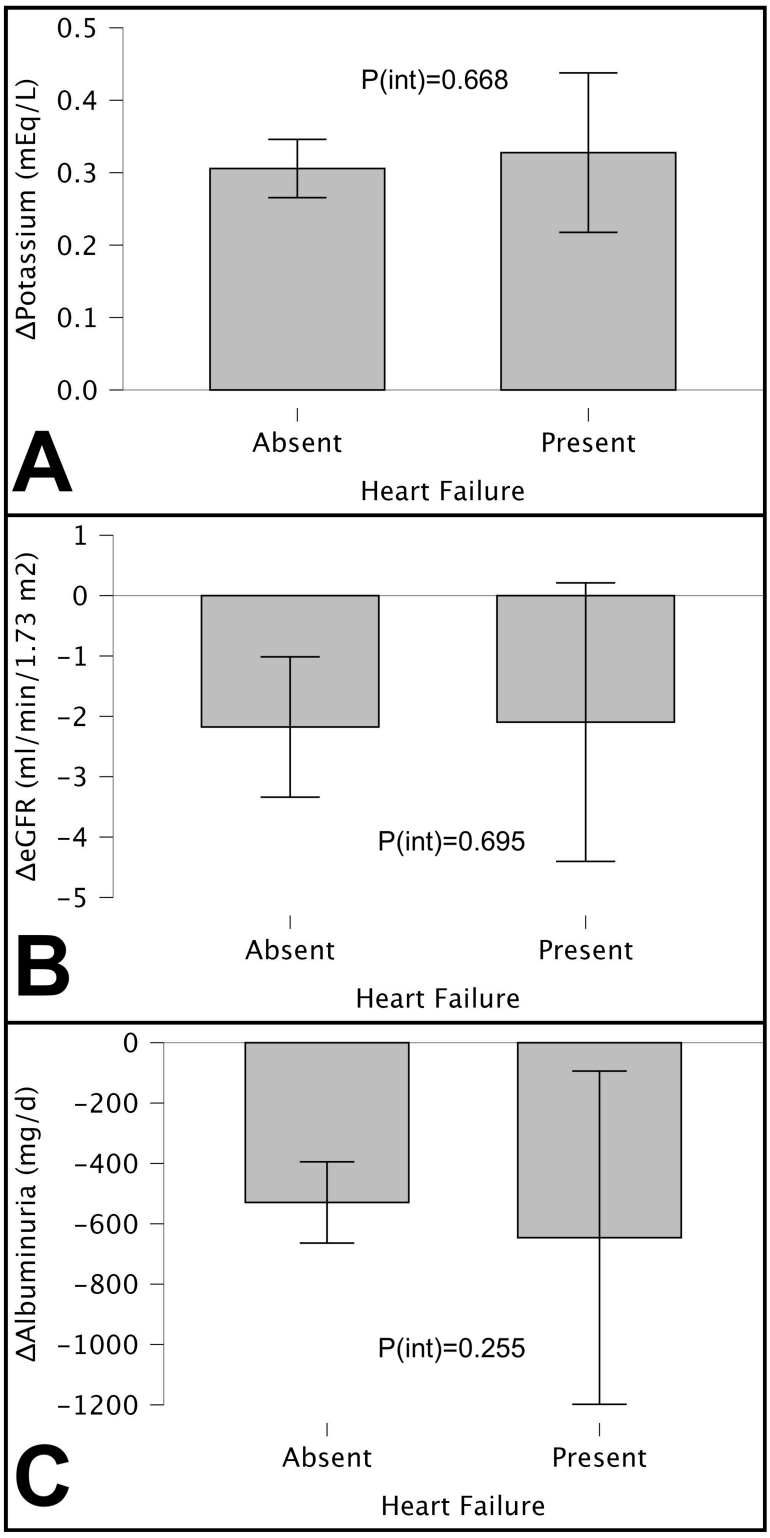

Figure 1

Bar charts showing the changes in potassium, estimated glomerular filtration rate and albuminuria in patients with and without heart failure. Lines show confidence intervals for means. P(int): P value for interaction.

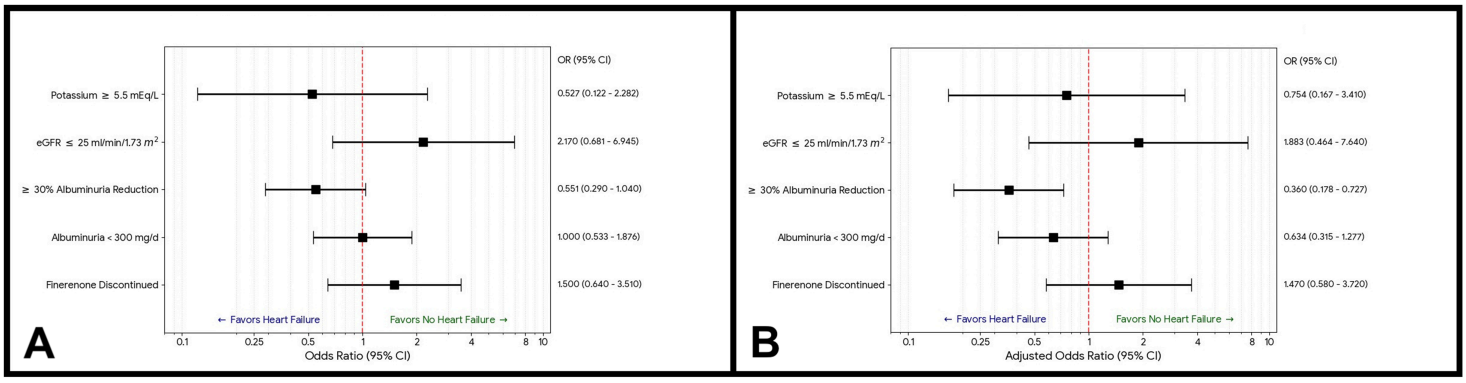

Figure 2

Forest plots showing individual safety and efficacy outcomes at 3rd month for unadjusted (A) and adjusted (B) analyses. eGFR: Estimated glomerular filtration rate, OR: Odds ratio, CI: Confidence interval.

Supplementary Files

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