Safety and Efficacy of Finerenone in Type 2 Diabetic Kidney Disease: A Meta-Analysis Postprint

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Abstract

Background Nonsteroidal mineralocorticoid receptor antagonist finerenone is a novel drug for renal protection in patients with diabetic kidney disease, and together with angiotensin-converting enzyme inhibitors and sodium-glucose cotransporter 2 inhibitors, constitutes renal protective agents for diabetic kidney disease patients. Recently, two meta-analyses in patients with chronic kidney disease regarding the effect of finerenone on estimated glomerular filtration rate decline have reached completely opposite conclusions. In view of this, this study specifically focused on patients with type 2 diabetes mellitus, aiming to thoroughly investigate the efficacy and safety of finerenone.

Objective To systematically evaluate the efficacy and safety of finerenone in patients with type 2 diabetic kidney disease.

Methods Computerized searches were conducted in Cochrane, Web of Science, Embase, and PubMed databases from inception to April 2024. Literature was screened and data extracted according to inclusion and exclusion criteria, and meta-analysis was performed using RevMan 5.3 to compare indicators such as urine albumin-to-creatinine ratio and estimated glomerular filtration rate in type 2 diabetes patients treated with finerenone.

Results A total of 7 articles comprising 15,528 patients were ultimately included. The results showed that compared with the control group, the intervention group (using finerenone) exhibited statistically significant differences in urine albumin-to-creatinine ratio (SMD=-0.46, 95%CI=-0.48~-0.39, P<0.05), estimated glomerular filtration rate (SMD=-0.15, 95%CI=-0.19~-0.10, P<0.05), kidney composite endpoint (OR=0.83, 95%CI=0.75~0.92, P<0.05), all-cause mortality (OR=0.88, 95%CI=0.78~0.99, P<0.05), and end-stage renal disease (OR=0.88, 95%CI=0.78~0.99, P<0.05). Compared with the control group, the risk of hyperkalemia was significantly increased in the intervention group (OR=2.13, 95%CI=1.89~2.39, P<0.05).

Conclusion Finerenone can significantly improve kidney composite endpoint events in patients with type 2 diabetic kidney disease, reduce urine albumin-to-creatinine ratio, and slow eGFR decline; the risk of hyperkalemia should be noted during treatment.

Full Text

Meta-Analysis of the Safety and Efficacy of Finerenone in the Treatment of Type 2 Diabetic Nephropathy

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Abstract

Background: Finerenone, a nonsteroidal mineralocorticoid antagonist, is a novel therapeutic agent for renal protection in patients with diabetic kidney disease, joining the ranks of angiotensin-converting enzyme inhibitors and sodium-glucose cotransporter 2 inhibitors in providing renal protection for such patients. Recently, two meta-analyses focusing on patients with chronic kidney disease have yielded conflicting conclusions regarding the impact of finerenone on the decline of estimated glomerular filtration rate (eGFR). In light of this, the present meta-analysis specifically targets the population with type 2 diabetes, aiming to thoroughly investigate the efficacy and safety of finerenone.

Objective: To systematically evaluate the efficacy and safety of finerenone in patients with type 2 diabetes and kidney disease.

Methods: A computerized search was conducted in the Cochrane, Web of Science, Embase, and PubMed databases, covering the period from their inception to April 2024. Literature was screened and data extracted according to the inclusion and exclusion criteria. Meta-analysis was performed using Revman 5.3, comparing indicators such as the urine albumin-to-creatinine ratio and estimated glomerular filtration rate in type 2 diabetes patients treated with finerenone.

Results: A total of 7 articles were ultimately included, involving 15,528 patients. The results showed that compared with the control group, the intervention group (using finerenone) had statistically significant differences in the urine albumin-to-creatinine ratio (SMD=-0.46, 95%CI=-0.48 to -0.39, P<0.05), estimated glomerular filtration rate (SMD=-0.15, 95%CI=-0.19 to -0.10, P<0.05), renal composite endpoint (OR=0.83, 95%CI=0.75 to 0.92, P<0.05), all-cause

mortality (OR=0.88, 95% CI=0.78 to 0.99, P<0.05), and end-stage renal disease (OR=0.88, 95%CI=0.78 to 0.99, P<0.05). Compared with the control group, the intervention group significantly increased the risk of hyperkalemia (OR=2.13, 95%CI=1.89 to 2.39, P<0.05).

Conclusion: Finerenone can significantly improve renal composite endpoint events in patients with type 2 diabetes and kidney disease, reduce the urine albumin-to-creatinine ratio, and slow down the decline of eGFR; however, attention should be paid to the risk of hyperkalemia during treatment.

Key words: Finerenone; Diabetic nephropathies; Safety; Efficacy; Meta-analysis

1. Materials and Methods

Diabetes is widely recognized as a major cause of chronic kidney disease (CKD), with approximately 60% of CKD cases worldwide developing from diabetes. Despite the implementation of multiple effective measures by health authorities to reduce risk factors associated with diabetic CKD, mortality rates from CKD and progression to end-stage renal disease (ESRD) continue to increase [1].

Renin-angiotensin system inhibitors (RASi) and sodium-glucose linked transporter-2 inhibitors (SGLT-2i) have been proven to slow the progression of diabetic nephropathy (DN) [2-3]. Additionally, mounting evidence indicates that overactivation of the mineralocorticoid receptor (MR) is a key factor promoting CKD development [4]. Recent evidence-based medical findings further confirm that mineralocorticoid receptor antagonists (MRA) can effectively delay CKD progression in patients with type 2 diabetes [5]. Nevertheless, traditional steroidal MRAs such as spironolactone and eplerenone have not been fully studied or widely accepted in CKD treatment due to their potential for sex hormone-related side effects and increased risk of hyperkalemia. In contrast, nonsteroidal MRAs like finerenone demonstrate higher receptor selectivity, effectively reducing sex hormone-related adverse events and lowering hyperkalemia risk [6]. Finerenone is distributed not only in the kidneys but also in cardiac tissue, providing dual cardioprotective and renoprotective effects by simultaneously inhibiting MR overactivation-induced inflammation and fibrosis in both organs [7].

Previous meta-analyses have examined finerenone versus placebo for treating kidney disease, including effects on urine albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) [8-9]. These studies yielded consistent results regarding improvement in renal indicators but varied in their findings on slowing eGFR decline. Notably, PITT et al.' s study [10] did not include patients with type 2 diabetes. Recent meta-analyses indicate that finerenone can significantly reduce UACR, slow eGFR decline, and maintain a favorable safety profile. However, a common limitation of these studies is the limited

number of included trials. The purpose of this study is to provide a more comprehensive evaluation of finerenone' s efficacy and safety in DN patients, aiming to offer a more robust evidence-based foundation for clinical practice.

1.1 Inclusion and Exclusion Criteria

Inclusion criteria: (1) Study population comprised patients with type 2 diabetes and kidney disease; (2) Intervention group received finerenone treatment; (3) Study type was published randomized controlled trials of finerenone treatment in DN patients; (4) Efficacy outcome indicators included changes in UACR and eGFR from baseline to follow-up; (5) Safety outcome indicators included renal composite endpoint (renal failure, kidney-related death, eGFR decline $ $40^{\{-1\}\cdot(1.73m}\{2\})\hat{}\{-1\}$], hyperkalemia (serum potassium $ $5.5 mmol/L), all-cause mortality, and ESRD (initiation of renal replacement therapy, kidney transplantation, serum creatinine >6.0 mg/dL, or renal failure).

Exclusion criteria: (1) Involving type 1 diabetes population; (2) Missing data that could not be obtained even after contacting the original authors.

1.2 Literature Search Strategy

A systematic search was conducted in Web of Science, PubMed, Cochrane, and Embase databases from inception to April 2024. Search keywords included "Finerenone," "kerendia," "BAY 94-8862," "Chronic Renal Insufficiencies," "Renal Insufficiencies, Chronic," "randomized controlled trials," "placebos," and "random allocation."

1.3 Literature Screening and Data Extraction

First, titles and abstracts of all retrieved literature were independently reviewed to exclude studies that clearly did not meet inclusion criteria. Subsequently, full texts of potentially eligible studies were read to confirm eligibility. During data extraction, a self-designed form was used to systematically record relevant information, including: (1) Basic study information: trial NCT number, author names, publication date; (2) Patient characteristics: gender, age, sex ratio; (3) Study outcome measures: UACR, eGFR, and renal composite endpoints.

1.4 Statistical Analysis

This meta-analysis was performed using Revman 5.4 software. Statistical results were described using odds ratios (OR), standardized mean differences (SMD), and 95% confidence intervals. Specifically, SMD was used as the effect size for continuous variables, while OR was used for dichotomous variables. To assess heterogeneity among studies, the ^{2} test (significance level α=0.1) was used, combined with I^2 to quantify heterogeneity magnitude. If I^2<50% and P>0.1, heterogeneity was considered acceptable. Conversely, if P$ $0.1 and I\hat{}\{2\}$$ $50%,

significant heterogeneity was indicated, requiring further exploration of potential sources.

2. Results

2.1 Literature Screening Process and Results

A total of 184 articles were retrieved, with 7 articles ultimately included [11-17] (Figure 1 [Figure 1: see original paper]).

2.2 Characteristics of Included Studies

The 7 included studies comprised 15,528 patients with study durations ranging from 4 to 163 weeks. Mean patient age ranged from 62.9 to 73.1 years, mean UACR values from 18.8 to 851.9 mg/g, and eGFR from 42.3 to 67.8 $mL \cdot min^{-1} \cdot (1.73\ m^2)^{-1}$. Detailed information is provided in Table 1 .

2.3 Efficacy Outcomes

2.3.1 UACR Five studies reported UACR outcomes. Heterogeneity test results showed I^2=52%, indicating moderate heterogeneity. Meta-analysis results demonstrated that UACR levels in the intervention group were significantly lower than in the control group (SMD=-0.46, 95%CI=-0.49 to -0.42, P<0.05), as shown in Figure 2 [Figure 2: see original paper].

2.3.2 eGFR Six studies reported eGFR outcomes. Heterogeneity test results showed I^2=68%, indicating moderate heterogeneity. Meta-analysis results demonstrated that the rate of eGFR decline in the intervention group was significantly slower than in the control group (SMD=-0.15, 95%CI=-0.19 to -0.10, P<0.05), as shown in Figure 3 [Figure 3: see original paper].

2.3.3 Renal Composite Endpoint Two studies reported renal composite endpoint outcomes. Heterogeneity test results showed I^2=0%, indicating no heterogeneity. Meta-analysis results demonstrated that finerenone significantly reduced the risk of renal composite endpoint events compared with the control group (OR=0.83, 95%CI=0.75 to 0.92, P<0.05), as shown in Figure 4 [Figure 4: see original paper].

2.4 Safety Outcomes

2.4.1 Acute Kidney Injury Six studies reported acute kidney injury outcomes. Heterogeneity test results showed I^2=0%, indicating no heterogeneity. Meta-analysis results showed no statistically significant difference in acute kidney injury risk between the intervention and control groups (OR=0.95, 95%CI=0.79 to 1.14, P=0.57), as shown in Figure 5 [Figure 5: see original paper].

2.4.2 Hyperkalemia Six studies reported hyperkalemia outcomes. Heterogeneity test results showed I^2=69%, indicating moderate heterogeneity. Meta-analysis results demonstrated that the risk of hyperkalemia events was significantly increased in the intervention group compared with the control group (OR=2.13, 95%CI=1.89 to 2.39, P<0.05), as shown in Figure 6 [Figure 6: see original paper].

2.4.3 All-Cause Mortality Four studies reported all-cause mortality outcomes. Heterogeneity test results showed I^2=0%, indicating no heterogeneity. Meta-analysis results demonstrated that all-cause mortality was significantly lower in the intervention group compared with the control group (OR=0.88, 95%CI=0.78 to 0.99, P<0.05), as shown in Figure 7 [Figure 7: see original paper].

2.4.4 ESRD Two studies reported ESRD outcomes. Heterogeneity test results showed I^2=10%, indicating low heterogeneity. Meta-analysis results demonstrated that finerenone significantly reduced the risk of ESRD events compared with the control group (OR=0.88, 95%CI=0.78 to 0.99, P<0.05), as shown in Figure 8 [Figure 8: see original paper].

3. Discussion

The results of this study demonstrate that finerenone has significant efficacy in reducing UACR and renal composite endpoint events, which may be related to its anti-inflammatory and anti-fibrotic mechanisms of action [18]. However, in clinical application, attention must be paid to finerenone' s effect on eGFR reduction and its increased risk of hyperkalemia. Although finerenone showed positive effects in reducing all-cause mortality and ESRD risk, these findings were based on a limited number of studies and require further validation.

Research has found that in patients with stage 4 chronic kidney disease or earlier, aldosterone levels may increase up to four times the reference value, triggering inflammation and fibrosis in target organ tissues [19]. Given that MR is expressed in both kidneys and heart, MR antagonist intervention has become an important strategy for improving adverse cardiovascular and renal outcomes [20]. Finerenone, a representative MR antagonist, offers higher selectivity and lower risks of gynecomastia and hyperkalemia compared with traditional steroidal MRAs [21].

Studies have shown that changes in urinary albumin positively correlate with ESRD and mortality risk in DN patients [22]. This study found that compared with the control group, finerenone treatment significantly reduced UACR levels, ESRD, and all-cause mortality. Although our results showed slowed eGFR decline in the finerenone group, long-term follow-up data from the FIDELIO-DKD trial revealed deeper insights: the finerenone group showed slower eGFR decline after 4 months, and over approximately 26 months of observation, the

eGFR decline remained relatively small, suggesting potential long-term benefits in slowing renal failure progression [11]. Finerenone' s renoprotective effects are attributed to its multiple mechanisms of anti-inflammatory, anti-fibrotic, and antioxidant actions [23]. The drug promotes differentiation of M2 macrophages, which as anti-inflammatory immune cells can promote effective tissue repair and inhibit fibrosis development through IL-4 receptor signaling pathways [24]. Finerenone also demonstrates direct anti-fibrotic effects, reducing myofibroblast proliferation and collagen accumulation while downregulating PAI-1 and NKD2 expression in mouse kidney disease models [25]. Additionally, its antioxidant properties enhance NO bioavailability and increase SOD activity, thereby improving endothelial function [26]. These findings support the potential sustained renoprotective effects of finerenone in DN patients.

Regarding safety, the finerenone group did not show significantly increased acute kidney injury risk but did demonstrate a significantly increased hyperkalemia risk compared with the control group. Among the 7 included studies, background treatment for DN patients included RASi. While studies suggest that although steroidal MRAs may be limited in treating patients receiving RASi due to increased hyperkalemia risk [27], nonsteroidal MRA finerenone also shows independent association with hyperkalemia [28]. However, finerenone' s balanced distribution between heart and kidney in rodent models may help reduce electrolyte disturbances, including hyperkalemia risk, in clinical studies [7,16]. This perspective is based on finerenone's tissue distribution characteristics, suggesting potential advantages in maintaining electrolyte balance. Furthermore, pooled analysis of the FIDELIO-DKD and FIGARO-DKD trials indicated that treatment discontinuation due to hyperkalemia occurred in 1.7% of the finerenone group versus 0.6% in the placebo group. The FIGARO-DKD trial reported that serum potassium levels >5.5 mmol/L and >6.0 mmol/L occurred in 13.5% and 2.3% of finerenone patients respectively, compared with 2.3% and 1.2% in the placebo group, all within clinically acceptable ranges [29]. Despite increased hyperkalemia, the finerenone group showed significantly reduced all-cause mortality with no significant difference in acute kidney injury incidence. Therefore, through routine serum potassium monitoring and standardized hyperkalemia management, the potential impact of hyperkalemia can be mitigated, providing a scientific basis for finerenone' s clinical application.

This study has several limitations: (1) Some studies had relatively short follow-up periods, which may affect accurate assessment of finerenone' s efficacy; (2) Although the overall study size was large, the number of included independent trials was limited, with two studies having sample sizes of 72 and 96 (totaling 168/15,528, or 1.1% of the total population), which may limit generalizability; (3) Data collection for renal composite endpoint and ESRD events was limited to only two studies, indicating insufficient data that may affect in-depth analysis of these specific outcomes.

Finerenone offers a novel therapeutic strategy for DN through its unique mechanism of action. This meta-analysis demonstrates that finerenone significantly

reduces renal composite endpoint events, UACR levels, and slows eGFR decline in DN patients. However, the risk of hyperkalemia requires careful monitoring during treatment. Future research should examine finerenone' s effects in different patient populations (varying ages, sexes, ethnicities, and comorbidities) and its interactions with other medications. Additionally, long-term follow-up studies are crucial for evaluating finerenone' s long-term efficacy and safety. When considering finerenone for clinical treatment decisions, physicians should weigh its potential benefits against risks and provide individualized treatment recommendations.

Author Contributions: Adili·Tuersun was responsible for study design, data collection and organization, data analysis and interpretation, drafting the initial manuscript, controlling and revising the overall content, and played a core leading role in the implementation and output of this study. Cheng Gang played a key coordinating and guiding role in this study, responsible for formulating and optimizing the overall research plan to ensure scientific feasibility and direction. The corresponding author actively organized regular team discussions, promptly resolved issues encountered during the research, provided valuable academic guidance and suggestions to team members, and facilitated smooth progress of the study. Additionally, the corresponding author takes overall responsibility for the manuscript.

Conflict of Interest: This article has no conflicts of interest.

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Note: Figure translations are in progress. See original paper for figures.