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Finerenone as a therapeutic option for managing chronic kidney disease (CKD) linked to type 2 diabetes (T2D) and his other potential use. A summary of current research findings

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is a common, often late-diagnosed and progressive condition associated with increased morbidity and mortality, mainly due to elevated cardiovascular risk. Diabetes is the one of the leading causes of CKD. Due to the significant role that excessive mineralocorticoid receptor (MR) activation plays in the development of diabetic kidney disease (DKD), new therapies that target this pathway, are being investigated. Finerenone is a non-steroidal selective antagonist that reduces inflammation and fibrosis by blocking MR overactivity in the kidneys, heart, and blood vessels.

Materials and Methods: This review is based on a comprehensive analysis of studies on the use of finerenone in patients with CKD and type 2 diabetes (T2D). Additionally, this article explores the potential applications of finerenone. The review was developed using a PubMed database and ClinicalTrials.gov.

Results: The results of the FIDELIO-DKD and FIGARO-DKD trials, as well as the FIDELITY pooled analysis, showed that finerenone significantly improved renal and cardiovascular outcomes. They also showed that although hyperkalemia is a major adverse effect of finerenone treatment, the therapy is safe with appropiate monitoring and dose adjustment. In addition, its possible beneficial effects are in non-diabetic CKD, heart failure with preserved (HFpEF) and mildly reduced (HFmrEF) ejection fraction, advanced of CKD with lower estimated glomerular filtration rate (eGFR), and diabetic retinopathy.

Conclusion: Clinical studies have confirmed that finerenone is a valuable addition to nephroprotective and cardioprotective strategies in the management of CKD associated with T2D, as reflected in latest clinical guidelines. Its safety profile is generally acceptable. Ongoing clinical trials will help clarify whether the approved indications for finerenone can be extended to non-diabetic CKD or additional clinical settings.

Keywords: finerenone; chronic kidney disease (CKD); type 2 diabetes (T2D); diabetic kidney disease (DKD); mineralocorticoid receptor (MR);

1. Introduction

1.1 Chronic kidney disease (CKD)

Chronic kidney disease (CKD) is defined by Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group in 2024 as „[…] abnormalities of kidney structure or function, present for a minimum of 3 months, with implications for health." [1]

Based on findings from studies assessing the global prevalence, it is estimated that approximately 843,6 million people worldwide are affected by CKD. [2] In Poland, the estimated prevalence of CKD among adults is 5,8% (about 1,7 million individuals), which is lower than previously anticipated. [3]

The two most common etiologies of CKD are diabetes and hypertension. [3, 4] Other risk factors of CKD include recent or recurrent acute kidney injury (AKI) or acute kidney disease (AKD), obesity, cardiovascular disease, genitourinary disease (such as neprolithiasis), family history and genetic disorders affecting the kidneys (e.g. autosomal dominant polycystic kidney disease - ADPKD), smoking, alcohol use, certain medications or radiation nephritis, systemic disease (such as systemic lupus erythematosus, vasculitis, HIV infection or multiple myeloma), gestational disorders (e.g. preterm birth and eclampsia) and occupational exposures (such as pesticides or mercury). [1, 5]

As CKD progresses, the kidneys gradually lose their ability to excrete uremic toxins, regulate fluid, electrolyte, and acid-base balance, produce erythropoietin, convert vitamin D to its active form via 1-α-hydroxylation and control calcium and phosphate metabolism, leading to complications such as uremia, hypertension, volume overload, hyperkalemia, acidosis, mineral and bone disorder, and secondary hyperparathyroidism. [4] Most importantly, patients

with CKD have a significantly increased risk of cardiovascular events, especially in the advanced stages of CKD. [6]

CKD is frequently asymptomatic until the advanced stages (G4–G5); therefore, it is often detected incidentally through laboratory tests of serum creatinine, which are used to estimate GFR. In end-stage kidney disease (ESKD), patients may experience nonspecific symptoms like fatigue, nausea, poor appetite, lethargy, and skin itching. [5] During the subjective and physical examination, particular attention should be given to body weight, the skin (pallor due to anemia, pruritus from uremia), the cardiovascular system (blood pressure, dyspnea, edema), the nervous system (cognitive impairment, sleep disorders), the gastrointestinal system (metal taste in mouth, loss of appetite, nausea, or vomiting), and to changes in urine volume and characteristics (polyuria, oliguria, nocturia, foamy urine suggestive of proteinuria, or hematuria). [4]

The most commonly used equation for estimating GFR is Chronic Kidney Disease Epidemiology Collaboration formula (known as CKD-EPI 2021) based on measurement alone creatinine (eGFRcr), it is currently the recommended standard. Adding cystatin C to creatinine improves the accuracy of GFR estimation (eGFRcr-cys) and risk stratification in CKD, so it should be used when available. [1, 7, 8] Albuminuria, measured as the urine albumin-creatinine ratio (UACR), is used as a marker of kidney damage and indicates an increased progression risk of CKD. [9]

All patients with CKD, in addition to the tests assessing kidney function (serum creatinine, eGFR and UACR), should perform complete blood count, urinalysis, fasting blood glucose and glycated haemoglobin (if necessary oral glucose tolerance test), serum electrolytes, lipid profile, and ultrasonography (USG) of the urinary system. Renal USG should include evaluation of kidney size, echogenicity, corticomedullary differentiation, bilateral symmetry and assessment for structural abnormalities such as cysts, hydronephrosis, or nephrolithiasis. Meanwhile, duplex imaging helps assess blood flow and detect renal artery stenosis. If the clinical history suggests conditions such as lupus, multiple myeloma, vasculitis, viral infections (HBV, HCV, HIV) or genetic disorders, appropriate additional tests should be conducted to identify potential underlying causes of CKD. If the initial evaluation does not indicate the cause of CKD, a kidney biopsy should be considered to identify less common causes, such as glomerulonephritis or unexplained tubulointerstitial disease. [4, 5]

The comprehensive criteria for diagnosing CKD require the presence of either of the following for a minimum duration of three months: a decreased estimated eGFR of less than 60 ml/min/1,73 m² (corresponding to eGFR categories G3a to G5), or one or more markers of

kidney damage. These markers include albuminuria (UACR ≥30 mg/g), abnormalities in urine sediment, persistent hematuria, electrolyte and other disturbances linked to tubular disorders, histological alterations observed via kidney biopsy, structural abnormalities detected by imaging, or a history of kidney transplantation. [1] CKD is classified based on its etiology, GFR stage (G1–G5), and degree of albuminuria (A1–A3), which helps assess disease severity, risk of progression, prediction of potential complications and long-term outcomes. [1, 4, 5]

Table legend: 1 – low risk, no other signs of kidney disease – no CKD 2 – moderately increased risk 3 – high risk 4 – very high risk				Albuminuria categories		
				A1 (<30mg/g) normal to mildly increased	A2 (30-300mg/g) moderately increased	A3 (>300mg/g) severely increased
GFR categories (ml/min/1,73m²)	G1	normal or high	≥90	1	2	3
	G2	mildly decreased	60-89	1	2	3
	G3a	mildly to moderately decreased	45-59	2	3	4
	G3b	moderately to severely decreased	30-44	3	4	4
	G4	severely decreased	15-29	4	4	4
	G5	kidney failure	<15	4	4	4

Table 1: Classification – prognosis of CKD progression, according to KDIGO CKD Work Group 2024 [1]

The management of CKD involves causal treatment depending on etiology, inhibition of progression, prevention and treatment of comorbidities and complications, and preparation for renal replacement therapy, along with the initiation of such therapy when necessary. The treatment should focus on controlling modifiable predictors of chronic kidney disease progression, including hypertension, hyperglycemia, proteinuria, dyslipidemia, smoking, obesity, and the use of nephrotoxic agents. [1, 4, 5] Non-pharmacological strategies play an important role in slowing the progression of CKD and reducing cardiovascular risk, and include a low-salt, low-protein, plant-rich diet, regular physical activity, smoking cessation, and weight reduction in individuals with excessive body mass. Pharmacological nephroprotective therapies include the use of angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), sodium-glucose cotransporter 2 (SGLT2) inhibitors, and the new drug finerenone, a selective mineralocorticoid receptor antagonist. Additionally, if indicated, lipid-lowering, antihypertensive, and hypoglycemic therapies are employed to reduce cardiovascular risk. To manage complications, sodium bicarbonate is used for metabolic acidosis, potassium-binding agents such as patiromer for hyperkalemia, diuretics to control sodium and fluid balance, symptomatic therapies for conditions such as pruritus and sleep disturbances, as well as appropriate treatment for anaemia and bone and mineral disorders. Moreover, infections should be prevented by vaccinating against influenza,

pneumococcal disease, and hepatitis B, and nephrotoxic medications should be avoided. It is also crucial to adjust the dosages of renally eliminated drugs. When eGFR reaches stage G5 (<15 ml/min/1,73 m²), indicating end-stage kidney disease (ESKD), treatment options include renal replacement therapy (dialysis or kidney transplantation) or conservative (palliative) care. [4, 10]

To summary, CKD is a common, often late-diagnosed and progressive condition associated with increased morbidity and mortality, mainly due to elevated cardiovascular risk. Early diagnosis and appropriate managment are essential for the slowing of disease progression and the reduction of the risk of cardiovascular disease.

1.2 Diabetic kidney disease (DKD)

As noted above, diabetes is the leading cause of CKD, and diabetic kidney disease (DKD), a common microvascular complication affecting approximately 20-40% of patients with diabetes, is the most prevalent cause of end-stage kidney disease (ESKD) in the United States. [4, 11, 12] In type 2 diabetes, DKD may be present at diagnosis, whereas in type 1 diabetes, it typically develops 5–15 years after onset, which underlies the recommendation to initiate and continue once a year CKD screening (eGFR and UACR) at diagnosis for type 2 and 5 years post-diagnosis for type 1 diabetes. [11]

The diagnosis is based on the clinical presentation, which includes the presence of type 1 or type 2 diabetes mellitus, albuminuria and/or reduced eGFR, in the absence of other suspected causes of kidney damage. DKD should be considered if diabetic retinopathy is present, although this is not always the case. It is also possible for DKD to occur with normoalbuminuria. Other causes of kidney damage should be considered in the case of rapid changes in eGFR or albuminuria, absence of retinopathy in type 1 diabetes, or the presence of active urine sediment or nephrotic syndrome. In such cases, a kidney biopsy should be considered. [11]

The pathogenesis of DKD is due to chronic hyperglycaemia, which induces metabolic, haemodynamic, inflammatory, and fibrotic changes that progressively alter renal structure and function, clinically manifested by increased albuminuria and decreased GFR. These changes include elevated production of reactive oxygen species (ROS), infiltration and activation of immune cells in renal tissue, increased expression of pro-inflammatory cytokines and chemokines, and over-activation of pathways such as the mineralocorticoid receptor (MR), transforming growth factor β (TGF-β), protein kinase C (PKC), osteopontin (OPN) or advanced glycation end products (AGEs), together with intraglomerular hypertension. This

cascade contributes to glomerular hyperfiltration, enlargement of glomerular basement membrane (GBM) pores, inflammation, podocyte injury, myofibroblast infiltration, proliferation of mesangium cells, extracellular matrix accumulation with progressive renal glomerular sclerosis, tubular atrophy and interstitial fibrosis. The final stage in the evolution of DKD is renal fibrosis. Genetic and epigenetic factors also influence disease progression. Understanding the pathogenesis of diabetic kidney disease will enable the development of therapies that target specific molecular pathways to slow disease progression and reduce morbidity and mortality, particularly from increased cardiovascular risk, as patients with DKD are at elevated risk of heart failure, coronary heart disease, cardiac arrhythmias, and sudden cardiac death. [13] Due to the significant role that excessive mineralocorticoid receptor activation plays in the development of DKD, new therapies that target this pathway, such as finerenone, are being investigated.

1.3 Finerenone

Finerenone is a relatively new treatment option with a non-steroidal structure (unlike eplerenone and spironolactone, which have a steroidal structure) and acts as a potent, highly selective mineralocorticoid receptor antagonist. The drug was registered by the U.S. Food and Drug Administration (FDA) in July 2021 and by the European Medicines Agency (EMA) in March 2022. [14] In Poland, finerenone is indicated in adults (≥18 years old) for the treatment of chronic kidney disease (eGFR 15-60 ml/min/1,73 m^2 with albuminuria) associated with type 2 diabetes. The drug is available as 10 mg and 20 mg oral tablets, taken once daily. The starting dose is determined based on eGFR, with a target dose of 20 mg. In addition, serum potassium levels should be measured before starting and, depending on the level, monitored at the recommended frequency. [15]

As mentioned above, overactivation of the mineralocorticoid receptor (MR) contributes to the progression of CKD in diabetes. It is also suggested that this pathway plays a pathogenic role in non-diabetic CKD. The MR is expressed in both epithelial and non-epithelial tissues, including the kidney (collecting tubules, podocytes, myeloid cells, fibroblasts), blood vessels (endothelial cells, myeloid cells, fibroblasts, vascular smooth muscle cells) or heart (cardiomyocytes, fibroblasts, myeloid cells, endothelial cells, vascular smooth muscle cells, T lymphocytes). MR is activated by various factors such as aldosterone, cortisol, Rac family small guanosine triphosphatase 1 (Rac1), or hyperglycemia. Aldosterone, acts via the MR in the collecting tubules of the kidney, is an important regulator of water and sodium-potassium balance and is secreted in response to hyperkalemia, hyponatremia, or reduced intravascular

volume. Finerenone inhibits inflammation and fibrosis by blocking the overactivity of the mineralocorticoid receptor found in the kidneys, heart and blood vessels. Further clinical trials are needed to support the extension of mineralocorticoid receptor antagonist therapy beyond diabetic CKD. [16]

2. Materials and Methods

This review is based on a comprehensive analysis of randomized clinical trials, pooled data analyses, and observational studies on the use of finerenone in patients with chronic kidney disease (CKD) and type 2 diabetes (T2D). In addition, this article was realized based on a of current literature to evaluate the potential applications of finerenone beyond its approved indications. The review was developed using a PubMed database and ClinicalTrials.gov.

3. Results

3.1 The use of finerenone in treating patients with chronic kidney disease (CKD) and type 2 diabetes (T2D)

The renal and cardioprotective effects of finerenone were demonstrated in the multicentre clinical trials: The Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease (FIDELIO-DKD) and The Finerenone in Reducing CV Mortality and Morbidity in Diabetic Kidney Disease (FIGARO-DKD). [17, 18] In addition, a pooled analysis called FIDELITY was conducted based on the above two trials. [19] The use of finerenone in clinical practice is being monitored in the ongoing FINE-real study. [20, 21, 22]

FIDELIO-DKD

This was a randomized, double-blind, international study in which adult patients (≥18 years old) with CKD and T2D, treated with the maximum dose of an ACEI or ARB with no adverse effects, were assigned in a 1:1 ratio to receive finerenone or placebo. The study was designed to evaluate the efficacy of finerenone in reducing the progression of CKD, hence the primary composite outcome was renal failure, a sustained decline in eGFR of at least 40% or death from renal causes. The secondary composite outcome was the assessment of cardiovascular risk by observing death from cardiovascular causes, non-fatal myocardial infarction, non-fatal stroke or hospitalisation for heart failure. Over a median follow-up of 2,6 years, the finerenone group had a lower incidence of the primary outcome event compared to the placebo group (17,8% vs. 21,1%) (hazard ratio [HR], 0,82; 95% confidence interval [CI], 0,73 to 0,93; P = 0,001), as well as a lower incidence of the secondary outcome event (13% vs.

14,8%) (HR, 0,86; 95% CI, 0,75 to 0,99; P = 0,03). This shows that finerenone reduces the risk of CKD progression and cardiovascular events compared to placebo in patients with CKD and T2D. Hyperkalemia occurred in 15,8% of patients on finerenone compared with 7,8% on placebo. The finerenone group was more likely than the placebo group (2,3% vs 0,9%) to discontinue treatment due to hyperkalaemia. [17]

FIGARO-DKD

In adult patients (≥18 years old) with CKD and T2D, treated with renin-angiotensin system blockade at the maximum tolerated dose without adverse effects, this study investigated the impact of finerenone on reducing cardiovascular morbidity and mortality. The primary composite outcome was the assessment of cardiovascular risk by observing death from cardiovascular causes, non-fatal myocardial infarction, non-fatal stroke or hospitalisation for heart failure, while the secondary composite outcome was renal failure, a sustained decline in eGFR of at least 40% or death from renal causes. A total of 7437 patients were enrolled based on one of two inclusion criteria: either moderately increased UACR (30-300 mg/g) with an eGFR of 25-90 ml/min/1.73 m² (stage 2 to 4 CKD) or severely increased UACR (300-5000 mg/g) with an eGFR ≥60 ml/min/1.73 m² (stage 1 or 2 CKD). Over a median follow-up of 3,4 years, the finerenone group had a lower incidence of the primary outcome event compared to the placebo group (12,4% vs. 14,2%) (HR, 0,87; 95% CI, 0,76 to 0,98; P = 0,03), as well as a lower incidence of the secondary outcome event (9,5% vs. 10,8%) (HR, 0,87; 95% CI, 0,76 to 1,01). The difference in the primary composite outcome in favour of the finerenone group was mainly due to a lower rate of hospitalisation for heart failure. These data show that finerenone reduces the risk of cardiovascular events in patients with CKD and T2D in comparison to placebo. Hyperkalemia occurred in 10,8% of patients on finerenone compared with 5,3% on placebo. Treatment discontinuation due to hyperkalemia was more common in the finerenone group than in the placebo group (1,2% vs. 0,4%). [18]

FIDELITY

A pooled analysis of FIDELITY using data from the above two, FIDELIO-DKD and FIGARO-DKD studies, confirmed that finerenone had reduced the risk of chronic kidney disease progression and risk of cardiovascular events in a broad group of patients with CKD and T2D. The analysis included 13026 patients, and the composite cardiovascular outcome was lower in the finerenone group compared with the placebo group (12,7% vs. 14,4%) (HR, 0,86; 95% CI, 0,78 to 0,95, P = 0,0018), as was the composite renal outcome (5,5% vs. 7,1%)

(HR, 0,77; 95% CI, 0,67 to 0,88, P = 0,0002), resulting in a relative risk reduction of 14% for the composite cardiovascular outcome and 23% for the composite renal outcome. [19]

FINE-REAL (NCT05348733)

This is an international, observational, prospective study has started in 2022 to learn more about the use and safety of finerenone in clinical practice. The end of the study is planned for 2028.

The first analysis, based on data from 504 patients, shows that 92,3% remained on finerenone treatment at a median follow-up of 7 months. Finerenone therapy was mainly initiated in patients with very high (48,2%) and high (27,9%) risk according to the KDIGO risk categories. The starting dose was much more often 10 mg (87,9%). Adverse events were reported in 21,8% of participants, including hyperkalemia in 5%. Notably, there were no cases of hyperkalemia leading to hospitalisation, dialysis or death. [20, 21, 22]

3.2 Safety of finerenone regarding hyperkalaemia

As mentioned above, the most common adverse effect of finerenone is hyperkalemia, which is due to its action via mineralocorticoid receptor blockade. Finerenone has a lower risk of hyperkalemia than spironolactone, as confirmed by the Mineralocorticoid Receptor Antagonist Tolerability Study (ARTS). [23]

Based on data from the FIDELITY pooled analysis, a new model for the risk of new-onset hyperkalemia (serum potassium level >5,5 mmol/L) in patients with CKD and T2D has been created. It takes into account seven variable characteristics, including serum potassium >4,5 mmol/L, history of hyperkalemia, UACR >1000mg/g, haemoglobin <12 g/dl, absence of thiazide diuretics and absence of SGLT2 inhibitors. The maximum score is 12 points, with 0-3 points for low risk, 4-6 points for intermediate risk and 7-12 points for high risk. Its use may help to identify patients using finerenone who need additional interventions to reduce the risk of hyperkalemia. [24]

3.3 Other potential applications of finerenone

Ongoing or completed clinical trials are investigating the use of finerenone in patient populations with other comorbidities or diseases. These include people with CKD without diabetes (FIND–CKD, NCT05047263) [25, 26], CKD with type 1 diabetes (T1D) (FINE-ONE, NCT05901831) [27, 28], symptomatic heart failure (HF) with a preserved (HFpEF) or mildly reduced (HFmrEF) ejection fraction (FINEARTS-HF, NCT04435626) [29, 30], with

CKD and proteinuria in children [(FIONA, NCT05196035) and (FIONA OLE, NCT05457283)] [31, 32, 33]. In addition, a study evaluating the safety and efficacy of dual finerenone therapy with sodium-glucose cotransporter-2 inhibitors (SGLT2i) in CKD with T2D has been registered (CONFIDENCE study). [35, 36]

3.3.1 Finerenone as a potential drug for the treatment of CKD without diabetes

A retrospective study of 16 Chinese patients with CKD without diabetes who received finerenone showed a 44,52% reduction in UACR and a small, statistically insignificant increase in mean eGFR from 80,16 to 83,45 ml/min/1,73 m² over a 3-month follow-up period. [37]

FIND-CKD (Finerenone In Non-Diabetic Chronic Kidney Disease) (NCT05047263) is a ongoing multicentre, double-blind, placebo-controlled study in patients with CKD of non-diabetic etiology receiving a maximum tolerated dose of a renin-angiotensin system inhibitor. A total of 1584 patients were randomised to the study and results will include changes in eGFR over 32 months of follow-up and the composite cardiorenal outcome. Among these patients, chronic glomerulonephritis (57%, with most IgA nephropathy) and hypertensive/ischaemic nephropathy (29%) are the most common causes of CKD. [25, 26]

3.3.2 Finerenone as a potential drug for the treatment of symptomatic heart failure (HF) with a preserved (HFpEF) or mildly reduced (HFmrEF) ejection fraction

FINEARTS-HF (Finerenone trial to investigate efficacy and safety superior to placebo in patients with heart failure)

This was an international, double-blind, placebo-controlled, randomized trial, completed in 2024, to evaluate the effect of finerenone in patients with HFmrEF or HFpEF, in addition to optimal HF therapy. The finerenone group had an 18% lower relative risk of worsening HF events compared to the placebo group over a median follow-up of 32 months. A reduced proportion of patients dying from cardiovascular causes was also observed in the finerenone group (8,1% vs. 8,7%), but this difference was not statistically significant (HR, 0,93; 95% CI, 0,78 to 1,11). [29, 30]

FINE-HEART

This was a pooled analysis of the three randomised clinical trials mentioned above (FIDELIO-DKD, FIGARO-DKD and FINEARTS-HF), which evaluated the efficacy and safety of finerenone in patients with CKD, T2D and HF. Compared to placebo, finerenone significantly

lowered all-cause mortality (11,0% vs. 12,0%; HR 0,91, 95% CI: 0,84–0,99; P = 0,027), reduced the risk of HF–related hospitalization (HR 0,83; 95% CI: 0,75–0,92; P < 0,001), and decreased of a composite kidney outcome, including sustained eGFR decline and kidney failure (HR 0,80, 95% CI: 0,72–0,90; P < 0,001). The reduction in the incidence of cardiovascular death in the finerenone group did not reach statistical significance (4,4% vs. 5,0%; HR 0,89; 95% CI: 0,78-1,01; P = 0,076). [37]

3.3.3 Finerenone in patients with CKD and T2D with eGFR <25 ml/min/1,73 m²

This was a retrospective analysis of nine patients with CKD and T2D who received 10 mg of finerenone for six months and had a baseline eGFR of less than 25 ml/min/1,73 m² showed that the mean eGFR slope improved significantly from an initial mean slope of -7,63 ± 9,84 (ml/min/1,73 m²/year) to -1,44 ± 3,17 (ml/min/1,73 m²/6 months, P=0,038). The study showed that finerenone treatment was associated with a slower decline in eGFR, but did not significantly reduce proteinuria. Hyperkalemia was not observed. Further clinical trials involving larger patient populations are needed to determine the efficacy and safety of finerenone in this patient group. [38]

3.3.4 Finerenone as a potential drug to reduce progression of non-proliferative diabetic retinopathy (NPDR) in patients with CKD and T2D. ReFineDR (NCT04477707) and DeFineDR (NCT04795726).

These studies involved retrospectively collecting ophthalmic data from participants in the FIDELIO-DKD and FIGARO-DKD trials. For the study were qualify adults patients (≥18 years old) with CKD and T2D, receiving optimized renin–angiotensin system (RAS) blockade, and who had a routine eye exam confirming non-proliferative diabetic retinopathy (NPDR) within six months before or up to one month after randomization. The primary endpoint was the progression of NPDR, defined by the occurrence of vision-threatening complications (VTCs), including: development of anterior segment neovascularization, onset of diabetic macular edema (DME), progression to proliferative diabetic rethinopathy (PDR). Fewer finerenone-treated patients developed vision-threatening complications (VTCs) or required ophthalmic intervention than placebo-treated patients, hence this analysis suggests that finerenone may delay NPDR progression and reduce the need for ophthalmic interventions in patients with T2D and CKD. These findings require further investigation in a prospective, randomized study, due to the limitations of this study, to confirm the potential benefits of finerenone in this patient population. [39, 40, 41]

4. Conclusion

Chronic kidney disease (CKD), particularly in patients with type 2 diabetes (T2D), is a serious and widespread health problem. It contributes to increased morbidity, mortality, as well as higher healthcare costs. Therefore, early diagnosis and appropriate treatment are essential for the slowing of disease progression and the reduction of the risk of cardiovascular disease.

A new non-steroidal mineralocorticoid receptor inhibitor, finerenone, is a promising drug in patients with CKD and T2D. The results of the FIDELIO-DKD and FIGARO-DKD trials, as well as the FIDELITY pooled analysis, showed that finerenone significantly improved renal and cardiovascular outcomes compared to the placebo group. They also showed that although hyperkalemia is a major side effect of finerenone treatment, the therapy is safe with appropriate monitoring and adjustment. Preliminary data from the ongoing FINE-REAL study further confirm the tolerability and long-term use of finerenone in clinical practice. In addition, its beneficial effects are possible in non-diabetic CKD, heart failure with preserved (HFpEF) and mildly reduced (HFmrEF) ejection fraction, advanced of CKD with eGFR <25 ml/min/1,73m^2, and diabetic retinopathy.

In summary, finerenone represents a valuable addition to nephroprotective and cardioprotective strategies in the management of chronic kidney disease (CKD) associated with type 2 diabetes (T2D), as reflected in latest clinical guidelines. Ongoing clinical trials will help clarify whether the approved indications for finerenone can be extended to non-diabetic CKD or additional clinical settings.

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