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Kerendia® (Finerenone): Slowing the Progression of Chronic Kidney Disease in Patients with Type 2 Diabetes

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Chronic kidney disease (CKD) is categorized using six stages of estimated glomerular filtration rate (eGFR), and three proteinuria stages which are delineated using the urinary albumin-to-creatinine ratio (as seen in Table 1).[1] According to Eckardt et al, more than 10% of the population has CKD.[2] Prevalence increases with age, with ranges from 4% from 20-39 years, up to 47% in those 70 years or older in the United States.[2]

Widespread diseases such as hypertension and diabetes often contribute to the formation of CKD.[2] Chronic kidney disease and severe albuminuria are associated with decreased quality of life due to reduction in functional status.[3,4] In addition, reduced eGFR and albuminuria are independent risk factors associated with increased risk of cardiovascular disease.[5] An eGFR of <60 mL/min/1.73m^2 is associated with double the risk of heart failure, stroke, peripheral artery disease, atrial fibrillation, and coronary heart disease.[1] When eGFR is less than 30 mL/min/1.73m^2, left-ventricular hypertrophy can occur, and is thought to contribute to increased prevalence of sudden cardiac death.[1] In patients with CKD, 26% of mortality is contributed to sudden cardiac death, while the prevalence of sudden cardiac death is 6-13% in the general population.[1]

Studies have shown one way to slow the rate of CKD progression is to treat hypertension, which also has cardiovascular benefits.[1] Multiple medication classes are used in CKD for cardiovascular and blood pressure benefits. The Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommend a target systolic blood pressure of <120 mmHg in patients with CKD and high blood pressure, if tolerated, to reduce all-cause mortality and cardiovascular events.[3] Renin-angiotensin-system (RAS) inhibitors are considered first-line due to ability to lower blood pressure and albuminuria; thereby reducing kidney disease progression.[1,3] RAS inhibitors are recommended by the KDIGO guidelines in the following patients: those that have high blood pressure and CKD, with diabetes, and moderate (urine albumin-to-creatinine ratio (UACR) 30-300 mg/g) to severe albuminuria (UACR >300 mg/g), and in patients without diabetes with severe albuminuria.[3] RAS inhibitors are suggested in patients with high blood pressure, CKD, and moderately increased albuminuria without diabetes, and in patients without albuminuria or diabetes.[3] RAS inhibitors include angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs). ACE inhibitors work in the lungs and kidneys to inhibit angiotensin-converting enzymes to reduce angiotensin II production, while ARBs block angiotensin receptors to reduce the effect of angiotensin.[6]

In addition to RAS inhibitors, other antihypertensives may be utilized, but have limited data, within the CKD patient population.[3] Loop diuretics, such as furosemide, bumetanide, and torsemide are often also used to reduce fluid retention and blood pressure, however there is a lack of data on outcomes with use in CKD.[1] High doses of loop diuretics can lead to dehydration, which is a risk factor for acute kidney injury.[7,8] Beta blockers, when compared to both placebo and RAS inhibitors, have no added benefit in reducing CV (cardiovascular) outcomes or improving CKD in patients with CKD and severe albuminuria, with or without diabetes.[3] Mineralocorticoid receptor antagonists (MRA) such as spironolactone and eplerenone, can be used for refractory hypertension

Table 1 | KDIGO 2012 Guideline CKD Categorization[5]

GFR Categories (mL/min/1.73m^2) Description and Range		
G1	Normal/high	≥90
G2	Mildly decreased	60-89
G3a	Mildly to moderately decreased	45-59
G3b	Moderately to severely decreased	30-44
G4	Severely decreased	15-29
G5	Kidney failure	<15
Persistent Albuminuria Categories (mg/g) Description and Range		
A1	Normal to mildly increased	<30
A2	Moderately increased	30-300
A3	Severely increased	>300

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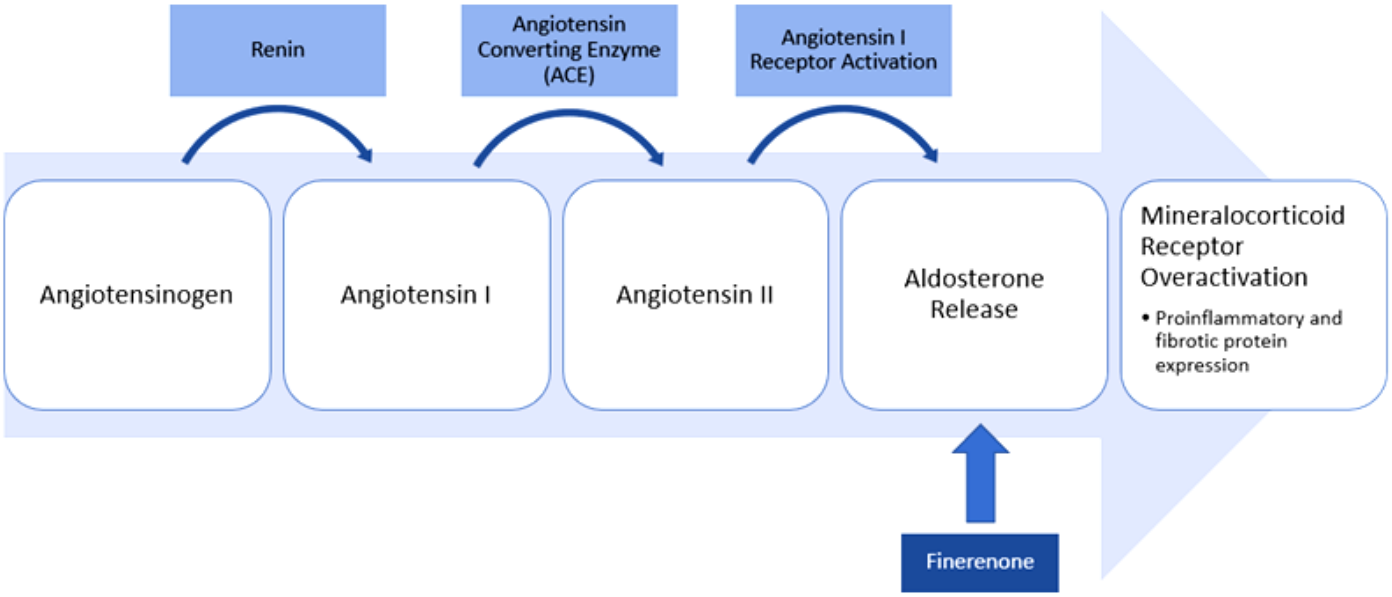
Figure 1 | RAAS Blockade & Finerenone Mechanism of Action[11,13]



in patients with CKD with average blood pressure lowering effect of 16 mmHg systolic and 9 mmHg diastolic (95% CI: 13-18 and 7 -10 mmHg; P<0.001).[3,9] In patients with diabetes and increased urinary albumin excretion, MRAs have been shown to lower albuminuria via aldosterone blockade that reduces prosclerotic growth.[3,10] However, some downfalls of MRAs include their non-specificity for the mineralocorticoid receptor leading to anti-androgenic side effects, like gynecomastia.[11] Therefore, MRAs are recommended in the KDIGO guidelines along with beta blockers only for resistant hypertension.[3]

Kerendia® (finerenone) approved by the Food and Drug Administration (FDA) in July 2021 is a selective MRA, developed by Bayer®, indicated for risk reduction of continued eGFR decline and to prevent end stage kidney disease, hospitalization for heart failure in patients with CKD associated with type 2 diabetes, cardiovascular death, and non-fatal myocardial infarction.[12] This paper will review finerenone pharmacology and characteristics along with important clinical trials leading to FDA approval.

Pharmacology

Mechanism of Action

Finerenone is a potent and selective nonsteroidal MRA that works within the RAAS pathway.[12,13] The RAAS pathway starts with renin release, renin cleaves angiotensinogen into angiotensin I, leading to conversion to angiotensin II.[11] This conversion is mediated by the angiotensin converting enzyme. Angiotensin II stimulates angiotensin I receptors which causes aldosterone release in the adrenal cortex.[11] Aldosterone then activates the mineralocorticoid receptor, increasing potassium secretion and sodium reabsorption (**Figure 1**).[11,13]

Overactivation of the mineralocorticoid receptor due to higher aldosterone release leads to pro-inflammatory and fibrotic protein expression.[13] These proteins cause fibrosis in the heart and kidneys which leads to organ damage.[13] Finerenone blocks the action of mineralocorticoid receptors by blocking aldosterone which in turn prevents the reabsorption of sodium and mineralocorticoid receptor overactivation in tissues such as the kidneys, heart, and blood vessels, and prevents fibrosis and inflammation.[11,12,13]

Pharmacodynamics

Finerenone is more selective for the mineralocorticoid receptor than spironolactone, which may decrease adverse events such as gynecomastia.[12] Finerenone also has a higher affinity for the mineralocorticoid receptor compared to eplerenone with an IC50 (50% inhibitory concentration) of 17.8 nM compared to eplerenone's IC50 of 990 nM.[11,12]

Pharmacokinetics

After oral administration, metabolism of finerenone results in an absolute bioavailability of 44%.[12] The maximum serum concentration of finerenone is reached 0.5 to 1.25 hours after the dose.[12] Finerenone volume of distribution at steady state is 52.6 L with 92% protein bound to serum albumin.[12] The terminal half-life is 2 to 3 hours.[12] Metabolism of finerenone into inactive metabolites occurs via CYP3A4 (90%) and CYP2C8 (10%).[12] Most of the dose is renally eliminated (80%) and the rest is eliminated in feces (20%). A summary of these pharmacokinetic properties can be seen in **Table 2.**[12]

Clinical Trials

Finerenone was approved by the FDA based on the FIDELIO-DKD (Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease) Trial which looked at whether finerenone decreases cardiovascular mortality and slows CKD progression in patients with CKD and diabetes. An additional trial, FIGARO-DKD (Finerenone in Reducing Cardiovascular Mortality and Morbidity in Diabetic Kidney Disease), consisted of patients with a broader range of kidney function than the FIDELIO-DKD Trial.

FIDELIO-DKD[14]

FIDELIO-DKD was a randomized, double-blind, and multicenter phase 3 trial that looked at the effect of finerenone on patients with CKD and type 2 diabetes compared to placebo.[14] The trial was conducted in adult patients with CKD associated with type 2 diabetes, who had a UACR of 30-300 mg/g, eGFR 25-60 mL/min/1.73m^2 and diabetic retinopathy, or UACR greater than 300 mg/g and an eGFR of 25-75 mL/min/1.73m^2.[14] Patients

were also required to be taking a max tolerated dose ACE inhibitor or ARB.[14] Serum potassium was required to be 4.8 mmol/L or less.[14] Exclusion criteria included reduced ejection fraction heart failure New York Heart Association class II-IV, a stroke, transient ischemic cerebral attack, acute coronary syndrome, or hospitalization for heart failure in the 30 days before the screening visit, A1c>12%, SBP≥170 mmHg or DBP≥110 mmHg at the run-in visit or SBP≥ 160 mmHg or DBP≥100 mmHg at the screening visit, or SBP<90 mmHg at either visit.[14]

The trial started with a run-in period where ACE inhibitors or ARBS were adjusted to max tolerated doses, after which eligibility was assessed.[14] Patients were assigned in a 1:1 ratio to either finerenone or placebo.[14] The dose of finerenone was renally adjusted, patients with an eGFR of 25-60 mL/min/1.73m^2 started with 10 mg once daily, while patients with an eGFR > 60 mL/min/1.73m^2 started with 20 mg once daily.[14] After one month, patients taking 10 mg were able to increase their dose to 20 mg once daily if serum potassium was 4.8 mmol/L or less and eGFR was stable.[14] Patients were also able to decrease their dose from 20 mg to 10 mg once daily at any time based on serum potassium.[14] If serum potassium was found to be greater than 5.5 mmol/L, then finerenone or the placebo were held.[14] The study treatment was restarted when potassium reached 5.0 mmol/L or lower.[14]

In total, 5,674 patients were included, with 2833 in the finerenone arm and 2841 in the placebo arm.[14] Baseline characteristics between the two groups were balanced.[14] The majority of patients studied were white men with an average age of 70 years.[14] The majority of patients were on a diuretic (56.6%), 28.5% were on a loop diuretic and 23.9% were on a thiazide diuretic, and 74.3% were on a statin.[14] The mean eGFR was 44.3±12.6 and the mean UACR was 852 (446-1634).[14] After 2.6 years, 29% of patients in the finerenone group and 28.2% of patients in the placebo did not complete the trial treatment, which was defined as having permanently discontinued the drug.[14] The most common reason for non-completion of the trial was due to an adverse outcome event, which was reported in 10.9% of patients in the finerenone arm and 10.3% of patients in the placebo arm.[13,14] Specifics on ADE with finerenone will be discussed later in this manuscript.

The primary composite endpoint (as seen in **Table 3**) was a sustained decline of eGFR of more than 40%, kidney failure, or death due to renal causes.[14] Finerenone was found to reduce the incidence of the primary composite endpoint, which occurred in 17.8% of patients in the finerenone group, compared to 21.1% of patients in the placebo group (HR 0.82, 95% CI 0.73-0.93, p=0.001) with a number needed to treat of 29 patients (95% CI, 16 to 166).[14] Finerenone was also found to reduce the secondary composite endpoint (as seen in **Table 4**) of CV death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for heart failure (HR 0.86, 95% CI 0.75-0.99, p=0.03).[14] The number needed to treat was 42 (95% CI, 22-397).[14] After 12 months, the mean systolic blood pressure was -2.1 mmHg with finerenone and 0.9 mmHg with placebo.[14]

Table 2 | Select Finerenone Pharmacokinetics[12]

Absorption	
T_{max}[a]	0.5—1.25 hours
Bioavailability	44%
Distribution	
V_{ss}[b]	52.6 L
Protein Binding	92%
Metabolism	
$T_{1/2}$[c]	2—3 hours
CYP3A4	90%
CYP2C8	10%
Elimination	
Fecal	20%
Urine	80%

[a]Time to maximum concentration; [b]Steady state volume of distribution; [c]Half-life

Given study results, the authors have concluded that patients taking finerenone had a lower risk of the primary composite outcome event which was a sustained decline of eGFR of more than 40%, kidney failure, or death due to renal causes, and a lower risk of secondary outcome events such as CV death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for heart failure.[14] This suggests that finerenone may be an effective treatment in patients with diabetes and CKD, for kidney and cardiovascular protection.[14]

FIGARO-DKD[15]

FIGARO-DKD is a randomized, double-blind, placebo-controlled, multi-center trial that looked at the effect of finerenone on patients with CKD and type 2 diabetes. This trial excluded patients with an UACR of 300 - 5000 and an eGFR of < 60 ml/minute/1.73 m^2 that were overrepresented in the FIDELIO-DKD trial.[15] Patients were randomly assigned to receive finerenone or placebo.[15] Investigators included adult patients that had type 2 diabetes who were taking a max tolerated dose of an ACE inhibitor or ARB. Patients must have been diagnosed with CKD which was defined as a UACR of 30 - 299 and eGFR of 25-90 ml/minute/1.73 m^2, or an UACR of 300 - 5000 and an eGFR of ≥ 60 ml/minute/1.73 m^2.[15] This trial had the same exclusion criteria and trial set up as the FIDELIO-DKD trial.[15] The trial started with a run-in period where RAS inhibitors were adjusted to the

Table 3 | Summary of Primary Outcomes[14,15]

Trial	Treatment Arms	Endpoint	Results (95% CI)	Hazard Ratio	P-Value
FIDELIO-DKD[14]	Finerenone (n=2,833)	Sustained decline of eGFR of more than 40%, kidney failure, or death due to renal causes	17.8 (0.73-0.93)	0.82	0.001
	Placebo (n=2,841)		21.1		
FIGARO-DKD[15]	Finerenone (n=3,686)	Death from cardiovascular causes, nonfatal myocardial infarction, nonfatal stroke, or hospitalization for heart failure	12.4 (0.76-0.98)	0.87	0.03
	Placebo (n=3,666)		14.2		

Table 4 | Summary of Secondary Outcomes[14,15]

Trial	Treatment Arms	Endpoint	Results (95% CI)	Hazard Ratio	P-Value
FIDELIO-DKD[14]	Finerenone (n=2,833)	CV death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for heart failure	13.0 (0.75-0.99)	0.86	0.03
	Placebo (n=2,841)		14.8		
FIGARO-DKD[15]	Finerenone (n=3,686)	First instance of kidney failure, an eGFR decrease of at least 40% for at least 4 weeks, or death due to renal causes	9.5 (0.76-1.01)	0.87	—
	Placebo (n=3,666)		10.8		

max tolerable dose, after which eligibility was assessed and patients were assigned to either finerenone or placebo and a 1:1 ratio.[15] The 10 mg or 20 mg dose of finerenone was assigned according to renal function and adjusted based on eGFR and serum potassium in the same way as the FIDELIO-DKD trial.[15]

In total, 7352 patients were included in the analysis with 3686 patients in the finerenone arm and 3666 patients in the placebo arm.[15] The different treatment arms were well balanced overall with the majority of patients in the study being white males with an average age of 64.1 years.[15] A majority of patients (70.5%) were taking statins while 47.6% of patients were taking diuretics.[15] The mean eGFR was 67.8±21.7 and the mean UACR was 308 (108-740).[15]

The primary composite outcome (as seen in **Table 3**) looked at death from CV causes, nonfatal myocardial infarction, nonfatal stroke, or hospitalization for heart failure.[15] The finerenone group showed a lower occurrence of the primary outcome compared to the placebo group, (12.4% vs 14.2%; HR: 0.87; 95% CI 0.76-0.98; P = 0.03).[15] The first secondary composite outcome (as seen in **Table 4**) looked at the first instance of kidney failure, an eGFR decrease of at least 40% for at least 4 weeks, or death due to renal causes.[15] This secondary outcome was non-significant, it occurred in 9.5% of patients in the finerenone group and 10.8% of patients in the placebo group (HR 0.87, 95% CI 0.76-1.01).15 After 24 months, the mean systolic blood pressure difference between finerenone and placebo was -2.6 mmHg.[15]

Based on the results of this study, the authors concluded that in patients with type 2 diabetes and stage 2 to 4 CKD with moderately elevated albuminuria, or stage 1 or 2 CKD with severely elevated albuminuria, patients in the finerenone group 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.[15] This suggests that finerenone would also be an effective treatment for kidney and cardiovascular protection for patients that were not included or underrepresented in the FIDELIO-DKD trial.[15]

Adverse Effects

Incidence of more common adverse events in the FIDELIO-DKD and FIGARO-DKD trials can be seen in the **Table 5**. In the FIDELIO-DKD trial, hyperkalemia was more common in the finerenone group (18.3%) compared to the placebo group (9.0%).[14] The same pattern was seen in the FIGARO-DKD trial, with an incidence of 10.8% in the finerenone arm and 5.3% in the placebo arm.[15]

In the FIDELIO-DKD and FIGARO-DKD trials, a serious adverse event was defined as an adverse event that resulted in death, hospitalization, was life threatening, caused a significant disability or birth defect, or the investigator determined it was a serious adverse event.[14] In the FIDELIO-DKD trial, a serious adverse event occurred in 31.9% of patients in the finerenone arm compared to 34.3% of patients in the placebo arm, but only a fraction of the serious adverse events were determined to be related to finerenone (1.7%) or placebo (1.2%).[14] In the FIGARO-DKD trial a serious adverse event occurred in 31.4% of patients in the finerenone arm and 33.2% of patients in the placebo arm.[15] However, a serious adverse event that was determined to be related to the trial regimen only occurred in 1.0% and 0.7% of patients respectively.[15]

Contraindications

As a CYP3A4 substrate, finerenone exposure is increased with concomitant use of CYP3A4 inhibitors.[12] Finerenone use is contraindicated with strong CYP3A4 inhibitors, such as itraconazole and clarithromycin, due to a large increase in finerenone exposure.[13] Increased finerenone exposure likely leads to hyperkalemia with unknown safety risks.[12] Concomitant use with a moderate or weak CYP3A4 inhibitor can lead to an increase in serum potassium and should be monitored at initiation and after dose changes.[12] The exposure of finerenone decreases with concomitant use of strong to moderate CYP3A4 inducers, which should be avoided.[12] Finerenone is also contraindicated in patients with adrenal insufficiency due to an increased risk of hyperkalemia.[16]

Dosage and Administration

Finerenone is available as a film-coated tablet in 10 mg and 20 mg strengths.[12] Starting dose is determined by kidney function and adjusted based on serum potassium levels. Finerenone can be taken with or without food and can be crushed and mixed with water or soft food. If a dose is missed, the dose should be taken as soon as possible on the same day, otherwise the dose should be skipped and the next dose taken as prescribed.[12]

After 4 weeks serum potassium should be measured and dosage adjusted appropriately.[12] If serum potassium is ≤ 4.8 then the dose should be either maintained at 20 mg once daily or increased to 20 mg once daily from 10 mg.[12] If serum potassium is 4.8 – 5.5 then the current dose should be maintained.[12] If serum potassium is >5.5 then finerenone should be withheld and restarted with previous maintenance dose once serum potassium is ≤5.[12] Serum potassium should be monitored 4 weeks after a dose adjustment.[12]

Special Populations

Renal Impairment

If eGFR is ≥60 then the recommended starting dose is 20

Table 5 | Common Adverse Effects with Finerenone[15]

Adverse Effect	Incidence Rate
Hyperkalemia	9.1—15.8%
Nasopharyngitis	8.5—8.6%
Arthralgia	5.0—8.1%
Anemia	5.9—7.4%

mg by mouth once daily, if eGFR is 25-60, then 10 mg by mouth once daily is recommended. If eGFR<25 then finerenone use is not recommended.

Hepatic Impairment

In patients with mild-moderate hepatic impairment (Child-Pugh Class A/B) dose adjustment of finerenone is not needed.[12] More frequent potassium monitoring in patients with Child-Pugh Class B hepatic impairment can be considered.[12] Patients with severe hepatic impairment (Child-Pugh Class C) should avoid finerenone use due to a lack of data.[12]

Pregnancy & Breastfeeding

There is no human data on the effect of finerenone on birth defects or adverse outcomes such as miscarriage.[12] At about four times the expected human exposure, animal studies in rats showed developmental toxicity.[12] Pup mortality increased and adverse effects such as a lower pup weight were seen.[12,13] However due to the lack of human data, there are no recommendations on finerenone use during pregnancy.[12]

Likewise, there is no data on if finerenone or metabolites are found in human breast milk or if finerenone effects breastfed infants.[12,13] However due to the developmental toxicity seen in rats it is likely found in rat milk, which means it is likely in human milk.[12,13] Therefore, breastfeeding is not recommended during treatment with finerenone and for one day after finerenone is stopped.[12,13]

Conclusion

Finerenone is a non-steroidal selective MRA that has been shown to be safe and effective for patients to reduce the risk of continued eGFR decline, end stage kidney disease, hospitalization for heart failure in patients with CKD associated with type 2 diabetes, cardiovascular death, and non-fatal myocardial infarction. Safety and efficacy were seen in clinical trials where finerenone was added to RAS inhibitors in patients with CKD A2 G2-G4 or CKD A3 G1 or G2, and type 2 diabetes. Current research is ongoing to determine use of finerenone in children, and safety and efficacy of finerenone in combination with empagliflozin. There are currently no trials being conducted to compare finerenone to other MRAs. The KDIGO guidelines recommend MRAs for refractory hypertension, however finerenone only had a small effect on blood pressure in clinical trials. Its place in guideline directed therapy is still not completely understood, but finerenone's effect on CKD outcomes shows it may have a place in therapy for those with CKD and type II diabetes.

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