Intended for HCPs in Scotland only.

Adverse event reporting information can be found below.
Prescribing Information for **Kerendia®▼** (finerenone) can be accessed via the QR code located at the bottom of this page.

Finerenone (Kerendia®▼)

For the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults

This promotional resource can be used as an information resource to support formulary applications

This formulary guide has been developed and funded by Bayer plc as an information source to aid completion of hospital formulary templates. Ideally, local hospital formulary templates should be completed using language and information that are relevant to local practice.

Reporting adverse events and quality complaints
▼ This medicine is subject to additional monitoring. This will allow quick identification of new safety information. Adverse events should be reported. Reporting forms and information can be found at https://yellowcard.mhra.gov.uk/. Adverse events should also be reported to Bayer plc. If you want to report an adverse event or quality complaint, reports can be directed to: Tel: 01182063500 or email: pvuk@bayer.com
Further information is available on the "contact" tab at www.bayer.co.uk.

Prescribing Information for Kerendia® (finerenone) is available via the QR code on the right.

Either click here or scan the QR for prescribing information.

For direct access to this prescribing information, please ensure that your device's browser settings have automatic PDF download enabled.

Date of preparation: November 2024 • Job bag code: PP-KER-GB-0745

Details of drug

Please refer to the Summary of Product Characteristics before prescribing Kerendia

Generic & brand name

Finerenone (Kerendia®▼)

Manufacturer

Bayer plc.

Licensed indication

Finerenone is indicated for the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults.[1]

Drug action

Finerenone is a nonsteroidal, selective antagonist of the mineralocorticoid receptor (MR). Evidence supports a pathophysiological role for overactivation of the MR in cardiorenal diseases through inflammation and fibrosis that lead to progressive kidney and cardiovascular dysfunction.[2]

Route of administration, pharmaceutical form & strengths available

Finerenone is to be administered orally with a glass of water, with or without food. Tablets should not be taken with grapefruit or grapefruit juice. The summary of product characteristics provides advice for patients who are unable to swallow tablets (sections 4.2 and 5.2).

Finerenone is available as film-coated tablets in 10 mg and 20 mg dosages.

Finerenone is available in packs of 28 tablets.

Posology

Treatment initiation

Estimated glomerular filtration rate (eGFR) and serum potassium have to be measured to determine if finerenone treatment can be initiated:

- In patients with an eGFR between ≥25 to <60 ml/min/1.73 m^2, finerenone may be initiated depending on their serum potassium (see below). According to the licensed indication, finerenone can be initiated in patients with CKD stage 3 and 4. This is equivalent to eGFR ≥15 to <60 ml/min/1.73 m^2. However, in patients with eGFR <25 ml/min/1.73 m^2, finerenone treatment should not be initiated due to limited clinical data.
- If serum potassium ≤4.8 mmol/l, finerenone treatment can be initiated.
- If serum potassium >4.8 to 5.0 mmol/l, initiation of finerenone treatment may be considered with additional serum potassium monitoring within the first 4 weeks based on patient characteristics and serum potassium levels.
- If serum potassium >5.0 mmol/l, finerenone treatment should not be initiated.

This is summarised in the figures below

eGFR (ml/min/1.73 m^2)	
<25	**≥25 to <60**
✘ Initiation of finerenone is not recommended	✔ Starting dose of finerenone: 10 mg once daily

Serum potassium levels	
mmol/l	**Initiation of finerenone**
≤4.8	✔ Can be initiated
>4.8 to 5.0	✔ May be considered*
>5.0	✘ Not recommended

*Initiation of finerenone may be considered with additional serum potassium monitoring within the first 4 weeks based on patient characteristics and serum potassium levels

- The starting dose is 10 mg finerenone once daily.
- The recommended target dose is 20 mg finerenone once daily.
- The maximum recommended dose is 20 mg finerenone once daily.
- No dose adjustment is necessary in elderly patients or based on body weight.
- Finerenone should not be initiated in patients with severe hepatic impairment as no data are available. No initial dose adjustment is needed in mild or moderate hepatic impairment but consider additional serum potassium monitoring in patients with moderate hepatic impairment and adapt monitoring according to patient characteristics.

Continuation of treatment

Serum potassium & eGFR have to be re-checked 4 weeks after:

- Initiation of treatment.
- An increase in dose to 20 mg once daily.
- Re-starting treatment (with 10 mg once daily).

Continuation of finerenone treatment and dose adjustment

	Current finerenone dose (once daily)	
Current serum potassium (mmol/l)	10 mg	20 mg
≤4.8	Increase to 20 mg finerenone once daily*	Maintain 20 mg once daily
>4.8 to 5.5	Maintain 10 mg once daily	Maintain 20 mg once daily
>5.5	Withhold finerenone Consider re-starting at 10 mg once daily when serum potassium ≤5.0 mmol/l	Withhold finerenone Re-start at 10 mg once daily when serum potassium ≤5.0 mmol/l

*Maintain 10 mg once daily, if eGFR has decreased >30% compared to the previous measurement

Thereafter, serum potassium should be re-measured periodically and as needed based on patient characteristics and serum potassium levels.

In patients with eGFR ≥15 ml/min/1.73 m^2, finerenone treatment can be continued with dose adjustment based on serum potassium. Due to limited clinical data, finerenone should be discontinued if patients have progressed to end-stage renal disease i.e. eGFR <15 ml/min/1.73 m^2.

Contraindications

- Hypersensitivity to the active substance or to any of the excipients (see section 6.1 of summary of product characteristics (SmPC)).
- Concomitant treatment with strong inhibitors of CYP3A4 e.g. itraconazole, ketoconazole, ritonavir, nelfinavir, cobicistat, clarithromycin, telithromycin, nefazodone (see section 4.5 of SmPC).
- Addison's disease.

Special warnings and precautions for use

Hyperkalaemia

Hyperkalaemia has been observed in patients treated with finerenone (see section 4.8 of SmPC).

Some patients are at a higher risk to develop hyperkalaemia.

Risk factors include low eGFR, higher serum potassium and previous episodes of hyperkalaemia. In these patients more frequent monitoring has to be considered.

Initiation and continuation of treatment (see section 4.2 of SmPC)

- If serum potassium >5.0 mmol/L, finerenone treatment should not be initiated.
- If serum potassium >4.8 to 5.0 mmol/L, initiation of finerenone treatment may be considered with additional serum potassium monitoring within the first 4 weeks based on patient characteristics and serum potassium levels.
- If serum potassium >5.5 mmol/L, finerenone treatment has to be withheld. Local guidelines for the management of hyperkalaemia have to be followed.
- Once serum potassium ≤5.0 mmol/L, finerenone treatment can be restarted at 10 mg once daily.

Monitoring

Serum potassium and eGFR have to be remeasured in all patients 4 weeks after initiation, re-start or increase in dose of finerenone. Thereafter, serum potassium has to be assessed periodically and as needed based on patient characteristics and serum potassium levels (see section 4.2 of SmPC).

Concomitant medications

The risk of hyperkalaemia also may increase with the intake of concomitant medications that may increase serum potassium (see section 4.5 of SmPC). See also 'Concomitant use of substances that affect finerenone exposure'.

Finerenone should not be given concomitantly with potassium-sparing diuretics (e.g., amiloride, triamterene) and other mineralocorticoid receptor antagonists (MRAs), e.g., eplerenone, esaxerenone, spironolactone, canrenone.

Finerenone should be used with caution and serum potassium should be monitored when taken concomitantly with potassium supplements, trimethoprim, or trimethoprim/sulfamethoxazole. Temporary discontinuation of finerenone may be necessary when patients have to take trimethoprim, or trimethoprim/sulfamethoxazole.

Renal impairment

The risk of hyperkalaemia increases with decreasing renal function. Ongoing monitoring of renal function should be performed as needed according to standard practice (see section 4.2 of SmPC).

Initiation of treatment

Finerenone treatment should not be initiated in patients with eGFR <25 mL/min/1.73 m^2 as clinical data are limited (see sections 4.2 and 5.2 of SmPC).

Continuation of treatment

Due to limited clinical data, finerenone treatment should be discontinued in patients who have progressed to end-stage renal disease (eGFR <15 mL/min/1.73 m^2).

Hepatic impairment

Finerenone treatment should not be initiated in patients with severe hepatic impairment (see section 4.2 of SmPC). These patients have not been studied (see section 5.2 of SmPC) but a significant increase in finerenone exposure is expected.

The use of finerenone in patients with moderate hepatic impairment may require additional monitoring due to an increase in finerenone exposure. Additional serum potassium monitoring and adaptation of monitoring have to be considered according to patient characteristics (see sections 4.2 and 5.2 of SmPC).

Heart failure

Patients with diagnosed heart failure with reduced ejection fraction and New York Heart Association II-IV were excluded from the phase III clinical study (see section 5.1 of SmPC).

Concomitant use of substances that affect finerenone exposure

Serum potassium should be monitored during concomitant use of finerenone with moderate or weak CYP3A4 inhibitors (see sections 4.2 and 4.5 of SmPC).

Finerenone should not be used concomitantly with strong or moderate CYP3A4 inducers (see section 4.5 of SmPC).

Grapefruit or grapefruit juice should not be consumed during finerenone treatment (see sections 4.2 and 4.5 of SmPC).

Embryo-foetal toxicity

Finerenone should not be used during pregnancy unless there has been careful consideration of the benefit for the mother and the risk to the foetus. If a woman becomes pregnant while taking finerenone, she should be informed of potential risks to the foetus.

Women of childbearing potential should be advised to use effective contraception during treatment with finerenone.

Women should be advised not to breast-feed during treatment with finerenone.

See sections 4.6 and 5.3 of SmPC for more information.

Information about excipients

Kerendia contains lactose

Patients with rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption should not take this medicinal product.

Kerendia contains sodium

This medicinal product contains less than 1 mmol sodium (23 mg) per tablet, that is to say essentially 'sodium-free'.

● Interaction with other medicinal products and other forms of interaction

Interaction studies have only been performed in adults.

Finerenone is cleared almost exclusively via cytochrome P450 (CYP)-mediated oxidative metabolism (mainly CYP3A4 [90%] with a small contribution of CYP2C8 [10%]).

Concomitant use contraindicated

Strong CYP3A4 inhibitors

Concomitant use of Kerendia with itraconazole, clarithromycin and other strong CYP3A4 inhibitors (e.g., ketoconazole, ritonavir, nelfinavir, cobicistat, telithromycin or nefazodone) is contraindicated (see section 4.3 of SmPC), since a marked increase in finerenone exposure is expected.

Concomitant use not recommended

Strong and moderate CYP3A4 inducers

Kerendia should not be used concomitantly with rifampicin and other strong CYP3A4 inducers (e.g., carbamazepine, phenytoin, phenobarbital, St John's Wort) or with efavirenz and other moderate CYP3A4 inducers. These CYP3A4 inducers are expected to markedly decrease finerenone plasma concentration and result in reduced therapeutic effect (see section 4.4 of SmPC).

Certain medicinal products that increase serum potassium

Kerendia should not be used concomitantly with potassium-sparing diuretics (e.g., amiloride, triamterene) and other MRAs (e.g., eplerenone, esaxerenone, spironolactone, canrenone). It is anticipated that these medicinal products increase the risk for hyperkalaemia (see section 4.4 of SmPC)

Grapefruit

Grapefruit or grapefruit juice should not be consumed during finerenone treatment, as it is expected to increase the plasma concentrations of finerenone through inhibition of CYP3A4 (see sections 4.2 and 4.4 of SmPC).

Concomitant use with precautions

Moderate CYP3A4 inhibitors

In a clinical study, concomitant use of erythromycin (500 mg three times a day) led to a 3.5-fold increase in finerenone AUC and 1.9-fold increase in its Cmax. In another clinical study, verapamil (240 mg controlled-release tablet once daily) led to a 2.7- and 2.2-fold increase in finerenone AUC and Cmax, respectively.

Serum potassium may increase, and therefore, monitoring of serum potassium is recommended, especially during initiation or changes to dosing of finerenone or the CYP3A4 inhibitor (see sections 4.2 and 4.4 of SmPC).

Weak CYP3A4 inhibitors

The physiologically-based pharmacokinetic simulations suggest that fluvoxamine (100 mg twice daily), increases finerenone AUC (1.6-fold) and Cmax (1.4-fold).

Serum potassium may increase, and therefore, monitoring of serum potassium is recommended, especially during initiation or changes to dosing of finerenone or the CYP3A4 inhibitor (see sections 4.2 and 4.4 of SmPC).

Certain medicinal products that increase serum potassium (see section 4.4 of SmPC).

Concomitant use of Kerendia with potassium supplements and trimethoprim, or trimethoprim/sulfamethoxazole is anticipated to increase the risk of hyperkalaemia. Monitoring of serum potassium is required.

Temporary discontinuation of Kerendia during trimethoprim, or trimethoprim/sulfamethoxazole treatment may be necessary.

Antihypertensive medicinal products

The risk for hypotension increases with concomitant use of multiple other antihypertensive medicinal products. In these patients, blood pressure monitoring is recommended.

Overview of the FIDELIO-DKD trial[1,2]

Design, patient population, study conduct and endpoints

FIDELIO-DKD was a double-blind, phase 3 trial which included 5,734 patients with chronic kidney disease (CKD) and type 2 diabetes (T2D) randomly assigned to receive finerenone or placebo. Eligible patients had a urinary albumin-to-creatinine ratio (with albumin measured in milligrams and creatinine measured in grams) of 30 to less than 300, an estimated glomerular filtration rate (eGFR) of 25 to less than 60 ml per minute per 1.73 m^2 of body surface area, and diabetic retinopathy, or they had a urinary albumin-to-creatinine ratio of 300 to 5000 and an eGFR of 25 to less than 75 ml per minute per 1.73 m^2 (see figure below).

Inclusion & exclusion criteria for FIDELIO-DKD

Inclusion criteria[1,2]

- T2D & CKD, defined as **either:**
  - UACR 30 to <300 mg/g & eGFR ≥25 to <60 ml/min/1.73 m^2* & a history of diabetic retinopathy **or**
  - UACR ≥300 to ≤5000 mg/g & eGFR ≥25 to <75 ml/min/1.73 m^2*
- Treated with either an ACEi or an ARB at maximal tolerated dose
- K+ ≤4.8 mmol/l

Key exclusion criteria[1,2]

- HFrEF with NYHA class II–IV
- Other kidney disease†
- Uncontrolled arterial hypertension‡
- Dialysis for acute kidney failure or kidney transplant

*Two recruitment caps were prespecified & closed per region:

1. The randomisation of patients with moderately increased albuminuria & diabetic retinopathy was restricted to ~10% of all randomised patients;

2. The randomisation of patients with severely increased albuminuria & eGFR 60–75 ml/min/1.73 m^2 was restricted to ~10% of all patients randomised with severely increased albuminuria;

†Known significant non-diabetic kidney disease, including clinically relevant renal artery stenosis;

‡Mean sitting SBP ≥170 mmHg or mean sitting DBP ≥110 mmHg at the run-in visit or mean sitting SBP ≥160 mmHg or mean sitting DBP ≥100 mmHg at the screening visit

Adapted from Bakris[2]

The trial consisted of run-in, screening, and double-blind treatment periods (see figure below). The run-in period allowed background medical therapies to be adjusted, including adjustment of ACE inhibitor or ARB therapy to a maximum labelled dose that did not cause unacceptable side effects. At the end of the run-in period, patients were reassessed for eligibility.

Eligible patients were then randomly assigned in a 1:1 ratio to receive oral finerenone or placebo; patients with an eGFR of 25 to less than 60 ml per minute per 1.73 m^2 at the screening visit received an initial dose of 10 mg once daily, and those with an eGFR of 60 ml per minute per 1.73 m^2 or more at the screening visit received an initial dose of 20 mg once daily. An increase in the dose from 10 to 20 mg once daily was encouraged after 1 month, provided the serum potassium level was 4.8 mmol per litre or less and the eGFR was stable; a decrease in the dose from 20 to 10 mg once daily was allowed any time after the initiation of finerenone or placebo. Patients in the placebo group underwent sham adjustment of the dose.

After randomisation, trial visits were conducted at month 1, month 4, then every 4 months until trial completion. Finerenone or placebo was withheld if potassium concentrations exceeded 5.5 mmol per litre and restarted when potassium levels fell to 5.0 mmol per litre or less.

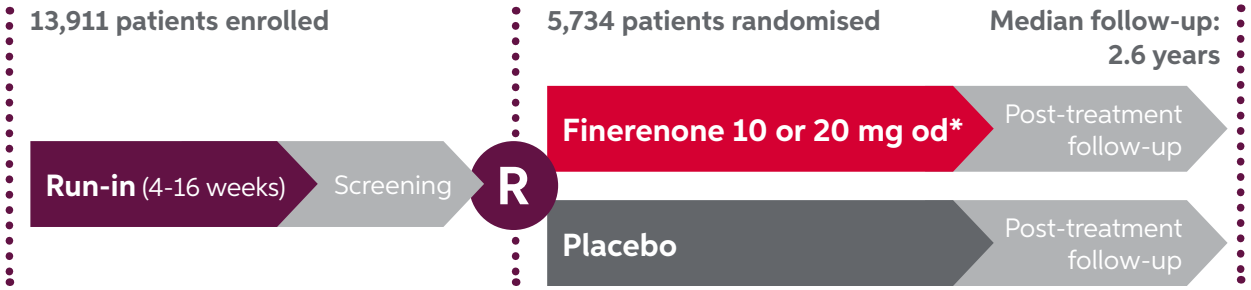
The primary composite outcome, assessed in a time-to-event analysis, was kidney failure, a sustained decrease of at least 40% in the eGFR from baseline, or death from renal causes. The key secondary composite outcome, also assessed in a time-to-event analysis, was death from cardiovascular causes, nonfatal myocardial infarction, nonfatal stroke, or hospitalisation for heart failure.

Other secondary outcomes (in order of sequential hierarchical testing) were death from any cause, hospitalisation for any cause, the change in the urinary albumin-to-creatinine ratio from baseline to month 4, and a composite of kidney failure, a sustained decrease of at least 57% in the eGFR from baseline (equivalent to a doubling of the serum creatinine level) maintained for at least 4 weeks, or death from renal causes (secondary composite kidney outcome).

A clinical event committee whose members were unaware of the trial-group assignments independently reviewed and adjudicated all reported outcome events.

FIDELIO-DKD study design

A randomised, double-blind, placebo-controlled, multicentre trial



Hierarchical endpoints

1. Kidney composite

- Time to kidney failure†
- Sustained ≥40% decrease in eGFR from baseline
- Death from renal causes

2. CV composite

- Time to CV death
- Non-fatal MI
- Non-fatal stroke
- Hospitalisation for HF

3. Death from any cause
4. Hospitalisation for any cause
5. Change in UACR
6. Second kidney composite‡

*10 mg if screening eGFR <60 ml/min/1.73 m^2; 20 mg if ≥60 ml/min/1.73 m^2, up-titration encouraged from month 1 if serum potassium ≤4.8 mEq/L & eGFR stable

†Kidney failure defined as end stage kidney disease (i.e. initiation of long-term dialysis for ≥90 days or kidney transplantation) or an eGFR of <15 ml/min/1.73 m^2

‡Secondary kidney composite defined as kidney failure, sustained decrease ≥57% in eGFR for ≥4 weeks or death from renal causes

Adapted from Bakris[2]

Safety analyses included assessment of adverse events and central laboratory testing; serum potassium and creatinine levels were also measured at local laboratories at all trial visits. Adverse events that occurred during the treatment period were defined as those that started or worsened during finerenone or placebo intake or up to 3 days after any temporary or permanent interruption.

Patient characteristics

Owing to critical Good Clinical Practice violations, 60 patients were prospectively excluded from all analyses, leaving 5,674 patients who were included in the statistical analyses. Baseline characteristics and concomitant medications were balanced between the two groups (see summary tables below with full details published in Bakris *et al*.[2]).

Baseline characteristics

Characteristic		Finerenone (n=2833)	Placebo (n=2841)	Total (n=5674)
Age, years, mean ± SD		65.4 ± 8.9	65.7 ± 9.2	65.6 ± 9.1
Male, n (%)		1953 (68.9)	2030 (71.5)	3983 (70.2)
HbA1c, %, mean ± SD		7.7 ± 1.3	7.7 ± 1.4	7.7 ± 1.3
Duration of T2D, years, mean ± SD		16.6 ± 8.8	16.6 ± 8.8	16.6 ± 8.8
BMI, kg/m^2, mean ± SD		31.1 ± 6.0	31.1 ± 6.0	31.1 ± 6.0
SBP, mmHg, mean ± SD		138.1 ± 14.3	138.0 ± 14.4	138.0 ± 14.4
DBP, mmHg, mean ± SD		75.8 ± 9.7	75.8 ± 9.7	75.8 ± 9.7
Smoking history, n (%)				
	Never smoked	1375 (48.5)	1371 (48.3)	2746 (48.4)
	Ex-smoker	1044 (36.9)	1078 (37.9)	2122 (37.4)
	Current smoker	414 (14.6)	392 (13.8)	806 (14.2)
Mean eGFR, ml/min/1.73 m^2, ± SD		44.4 ± 12.5	44.3 ± 12.6	44.3 ± 12.6
eGFR at baseline, ml/min/1.73 m^2, n (%)	<25	66 (2.3)	69 (2.4)	135 (2.4)
	25–<45	1476 (52.1)	1505 (53.0)	2981 (52.5)
	45–<60	972 (34.3)	928 (32.7)	1900 (33.5)
	≥60	318 (11.2)	338 (11.9)	656 (11.6)
Median UACR, mg/g, (IQR)		833 (441–1628)	867 (453–1645)	852 (446-1634)
UACR at baseline, mg/g, n (%)	<30	11 (0.4)	12 (0.4)	23 (0.4)
	30–<300	350 (12.4)	335 (11.8)	685 (12.1)
	≥300	2470 (87.2)	2493 (87.8)	4963 (87.5)
Mean serum potassium, mmol/l		4.37 ± 0.46	4.3 ± 0.46	4.37 ± 0.46

Medical history at baseline

Medical history, n (%)	Finerenone (n=2833)	Placebo (n=2841)	Total (n=5674)
Diabetic retinopathy	1312 (46.3)	1351 (47.6)	2663 (46.9)
Hypertension	2737 (96.6)	2768 (97.4)	5505 (97.0)
Diabetic neuropathy	738 (26.1)	716 (25.2)	1454 (25.6)
History of CV disease	1303 (46.0)	1302 (45.8)	2605 (45.9)
Coronary artery disease	842 (29.7)	860 (30.3)	1702 (30.0)
Myocardial infarction	378 (13.3)	388 (13.7)	766 (13.5)
Peripheral arterial occlusive disease	470 (16.6)	453 (15.9)	923 (16.3)
Ischaemic stroke	329 (11.6)	360 (12.7)	689 (12.1)
Heart failure	195 (6.9)	241 (8.5)	436 (7.7)

Baseline medications			
Medication use, n (%)	Finerenone (n=2833)	Placebo (n=2841)	Total (n=5674)
ACEis*	950 (33.5)	992 (34.9)	1942 (34.2)
ARBs*	1879 (66.3)	1846 (65.0)	3727 (65.7)
Alpha-blocking agents	693 (24.5)	715 (25.2)	1408 (24.8)
Beta blockers	1462 (51.6)	1506 (53.0)	2968 (52.3)
Diuretics	1577 (55.7)	1637 (57.6)	3214 (56.6)
Loop diuretics	786 (27.7)	833 (29.3)	1619 (28.5)
Thiazide diuretics	700 (24.7)	655 (23.1)	1355 (23.9)
Calcium antagonists	1773 (62.6)	1812 (63.8)	3585 (63.2)
Statins	2105 (74.3)	2110 (74.3)	4215 (74.3)
Potassium-lowering agents	70 (2.5)	66 (2.3)	136 (2.4)
Platelet aggregation inhibitors†	1633 (57.6)	1595 (56.1)	3228 (56.9)
Glucose-lowering therapies	2747 (97.0)	2777 (97.7)	5524 (97.4)
Insulin	1843 (65.1)	1794 (63.1)	3637 (64.1)
Metformin	1251 (44.2)	1239 (43.6)	2490 (43.9)
Sulfonylurea	654 (23.1)	673 (23.7)	1327 (23.4)
Alpha-glucosidase inhibitor	163 (5.8)	161 (5.7)	324 (5.7)
DPP-4is	764 (27.0)	758 (26.7)	1522 (26.8)
GLP-1RAs	189 (6.7)	205 (7.2)	394 (6.9)
SGLT-2is	124 (4.4)	135 (4.8)	259 (4.6)

*14 patients were not treated with an ACEi or ARB at baseline & 7 patients were treated with both an ACEi & an ARB

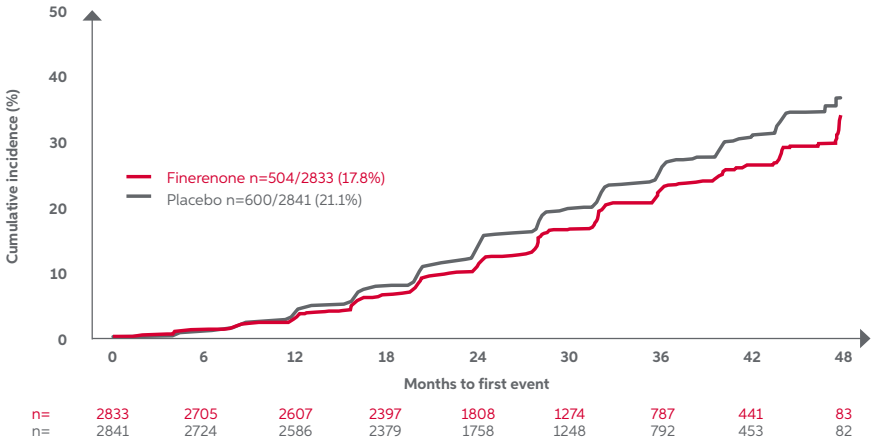
†Excluding heparins

Results – efficacy outcomes

The incidence of the primary composite outcome of kidney failure, a sustained decrease of at least 40% in the eGFR from baseline, or death from renal causes was significantly lower in the finerenone group than in the placebo group, occurring in 504 patients (17.8%) and 600 patients (21.1%), respectively (hazard ratio [HR], 0.82; 95% confidence interval [CI], 0.73 to 0.93; p=0.001).

The incidences of the components were all non-significant; most showed a numerical trend in favour of finerenone and the effects of finerenone on the primary outcome were generally consistent across prespecified subgroups.

Time to event analyses – primary composite outcome of kidney failure, a sustained decrease of at least 40% in the eGFR from baseline, or death from renal causes



*Defined as end-stage kidney disease (initiation of dialysis for ≥90 days or kidney transplantation), or an eGFR <15 ml/min/1.73 m² over ≥4 weeks

RRR: 18%[1]

HR: 0.82
95% CI: 0.73-0.93
p=0.001
Median follow-up: 2.6 years

At 36 months[1]

ARR: 3.4%	NNT: 29
95% CI: 0.6-6.2	95% CI: 16-166 (to prevent one primary outcome)

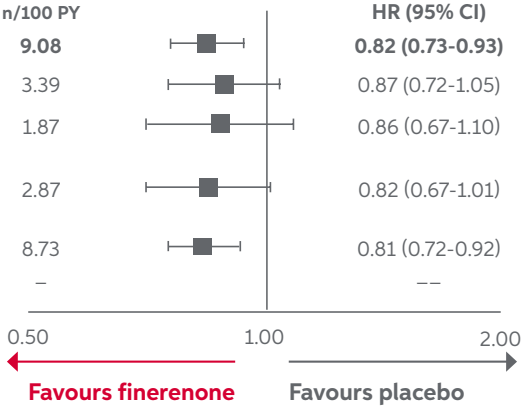
Lower risk of a primary composite renal outcome event:

- kidney failure*, or
- sustained decrease of ≥40% in eGFR from baseline, or
- death from renal causes

Adapted from Bakris[2]

Components of primary renal composite endpoint

Outcome	Finerenone n=2833 n (%)	Finerenone n=2833 n/100 PY	Placebo n=2841 n (%)	Placebo n=2841 n/100 PY	HR (95% CI)	p value
Primary composite outcome	**504 (17.8)**	**7.59**	**600 (21.1)**	**9.08**	**0.82 (0.73-0.93)**	**0.001**
Kidney failure*	208 (7.3)	2.99	235 (8.3)	3.39	0.87 (0.72-1.05)	––
End-stage kidney disease	119 (4.2)	1.60	139 (4.9)	1.87	0.86 (0.67-1.10)	––
Sustained† decrease in eGFR to <15 ml/min/1.73 m²	167 (5.9)	2.40	199 (7.0)	2.87	0.82 (0.67-1.01)	––
Sustained† decline of ≥40% in eGFR from baseline	479 (16.9)	7.21	577 (20.3)	8.73	0.81 (0.72-0.92)	––
Death from renal causes	2 (<0.1)	–	2 (<0.1)	–	––	––



Adapted from Bakris[2]

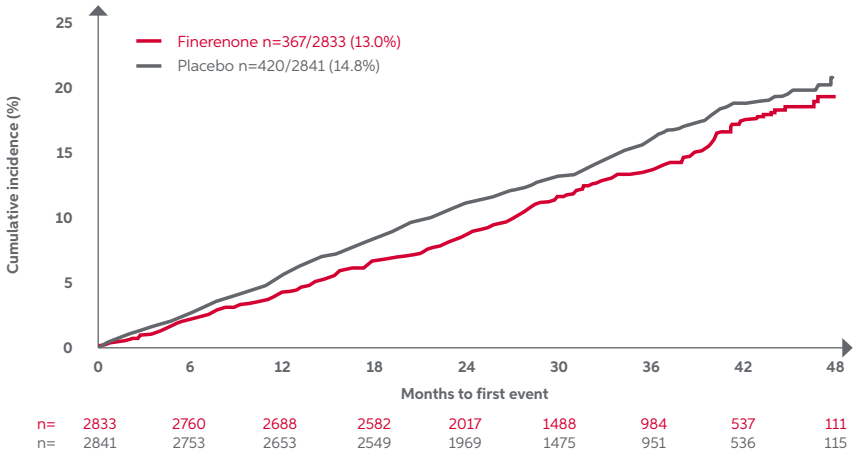
According to N Eng J Med policy, p-values are not reported for components of composite outcomes

*Kidney failure defined as either ESKD (initiation of chronic dialysis for ≥90 days or kidney transplant) or sustained decrease in eGFR <15 ml/min/1.73 m²

†confirmed by two eGFR measurements ≥4 weeks apart

Patients in the finerenone group also had a significantly lower risk of a key secondary outcome event (death from cardiovascular causes, nonfatal myocardial infarction, nonfatal stroke, or hospitalisation for heart failure), which occurred in 367 patients (13.0%) in the finerenone group and 420 patients (14.8%) in the placebo group (HR, 0.86; 95% CI, 0.75 to 0.99; p=0.03). The incidences of the components were all non-significant; most showed a numerical trend in favour of finerenone. The treatment effect for the key secondary endpoints was generally consistent across subgroups. Finerenone is not licensed for this indication. Finerenone is indicated for the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults.

Time to event analyses – secondary composite outcome of death from cardiovascular causes, non-fatal myocardial infarction, non-fatal stroke, or hospitalisation for heart failure



RRR: 14%[1]

HR: 0.86
95% CI: 0.75-0.99
p=0.03
Median follow-up: 2.6 years

ARR at 36 months: 2.4%
95% CI: 0.3-4.5

Risk of a secondary cardiovascular outcome event:[1]

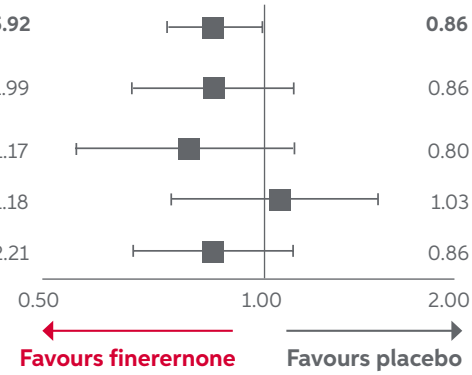
- death from CV causes, or
- non-fatal MI, or
- non-fatal stroke, or
- hospitalisation for heart failure

Adapted from Bakris[2]

Time to event analyses – secondary composite outcome of death from cardiovascular causes, non-fatal myocardial infarction, non-fatal stroke, or hospitalisation for heart failure.

Components of secondary CV composite endpoint

Outcome	Finerenone n=2833 n (%)	Finerenone n=2833 n/100 PY	Placebo n=2841 n (%)	Placebo n=2841 n/100 PY	HR (95% CI)	p value
Key secondary composite outcome	**367 (13.0)**	**5.11**	**420 (14.8)**	**5.92**	**0.86 (0.75-0.99)**	**0.03**
Death from cardiovascular causes	128 (4.5)	1.69	150 (5.3)	1.99	0.86 (0.68-1.08)	––
Non-fatal myocardial infarction	70 (2.5)	0.94	87 (3.1)	1.17	0.80 (0.58-1.09)	––
Non-fatal stroke	90 (3.2)	1.21	87 (3.1)	1.18	1.03 (0.76-1.38)	––
Hospitalisation for heart failure	139 (4.9)	1.89	162 (5.7)	2.21	0.86 (0.68-1.08)	––

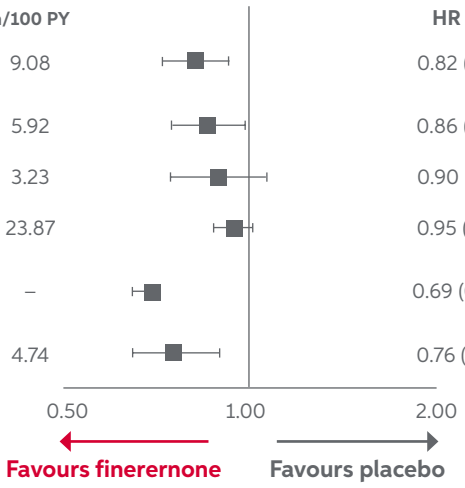


Adapted from Bakris[2]

There was no significant between-group difference in the risk of death from any cause; analyses of subsequent prespecified outcomes were, therefore, exploratory. This is summarised in the figure below.

Hierarchical endpoint analysis

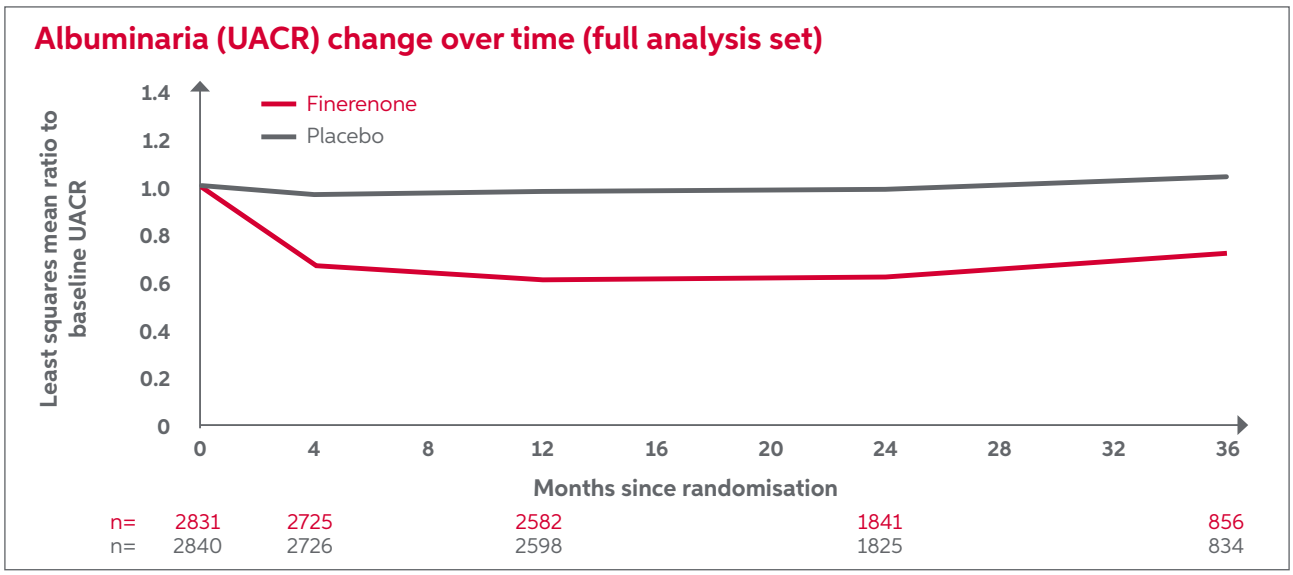
Outcome	Finerenone n=2833 n (%)	Finerenone n=2833 n/100 PY	Placebo n=2841 n (%)	Placebo n=2841 n/100 PY	HR (95% CI)	p value
Primary composite outcome	504 (17.8)	7.59	600 (21.1)	9.08	0.82 (0.73-0.93)	0.001
Key secondary composite outcome	367 (13.0)	5.11	420 (14.8)	5.92	0.86 (0.75-0.99)	0.03
Death from any cause	219 (7.7)	2.90	244 (8.6)	3.23	0.90 (0.75-1.07)	––
Hospitalisation for any cause	1263 (44.6)	22.56	1321 (46.5)	23.87	0.95 (0.88-1.02)	––
Change in UACR from baseline to month 4	–	–	–	–	0.69 (0.66-0.71)*	––
Secondary composite renal outcome*	252 (8.9)	3.64	326 (11.5)	4.74	0.76 (0.65-0.90)	––



Adapted from Bakris[2]

According to N Eng J Med policy p-values are only reported for hierarchical endpoints that reached statistical significance
*Ratio of least-squares mean (95% CI)

Finerenone was associated with a 31% greater reduction in the urinary albumin-to-creatinine ratio from baseline to month 4 than placebo (finerenone vs. placebo: mean change from baseline, -34.7% vs. -4.7%; ratio of least-squares mean change from baseline, 0.69; 95% CI, 0.66 to 0.71), and a lower mean urinary albumin-to-creatinine ratio with finerenone than with placebo was maintained thereafter.



Adapted from Bakris[2]

Results – safety outcomes[1,2]

The safety of finerenone was evaluated in 2,827 patients in the study who received finerenone (10 or 20 mg once daily), with a mean duration of treatment of 2.2 years.

The overall incidence of treatment-emergent adverse events was similar across the finerenone and placebo groups – summarised in the table below.

Investigator reported treatment-emergent AEs

Safety outcome, n (%)	Finerenone (n=2827)	Placebo (n=2831)
Any AE	2468 (87.3)	2478 (87.5)
AE related to study drug	646 (22.9)	449 (15.9)
AE leading to treatment discontinuation	207 (7.3)	168 (5.9)
Any serious AE	902 (31.9)	971 (34.3)
Serious AE related to study drug	48 (1.7)	34 (1.2)
Serious AE leading to treatment discontinuation	75 (2.7)	78 (2.8)

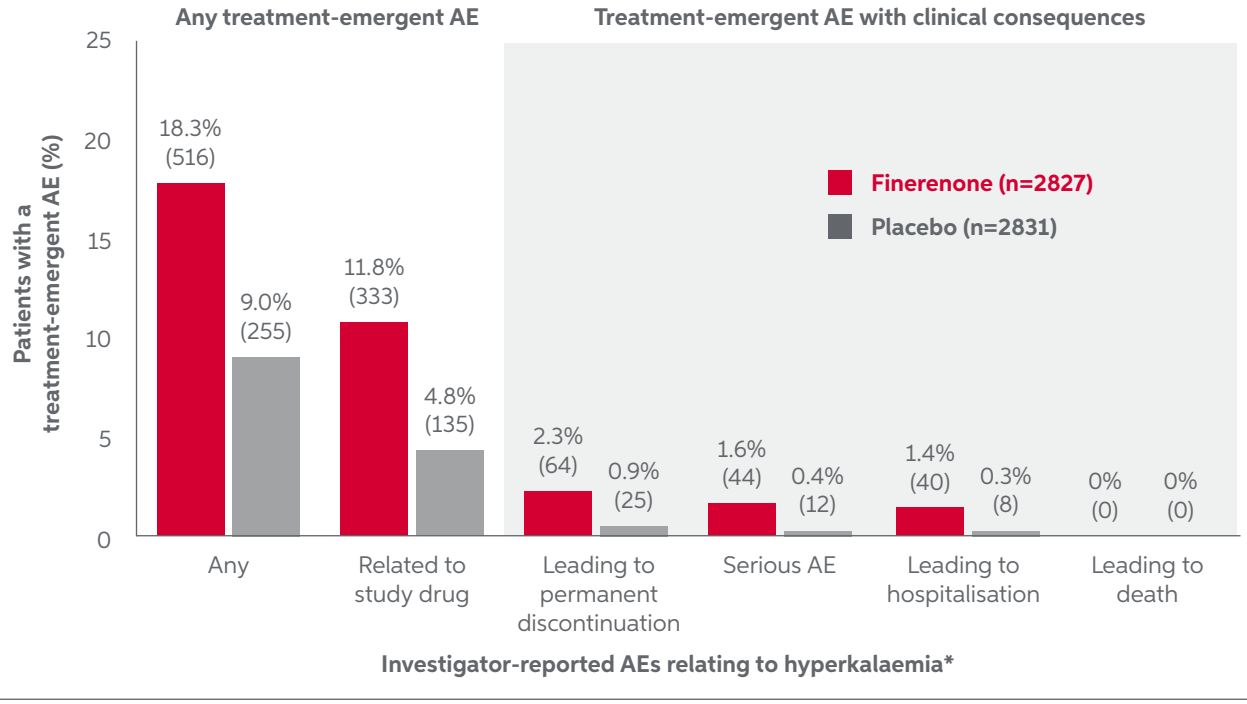
The most frequently reported adverse reaction under treatment with finerenone was hyperkalaemia. The other adverse reactions occurring in ≥1% of patients were hyponatraemia, hyperuricaemia, hypotension, pruritus and decreased GFR. Hyperuricaemia has been added to the SmPC as of July 2023 as a common AE.[1]

MedDRA system organ class	Very common (≥10%)	Common (≥1% to <10%)	Uncommon (≥0.1% to <1%)
Metabolism & nutrition disorders	Hyperkalaemia	Hyponatraemia Hyperuricaemia	
Vascular disorders		Hypotension	
Skin & subcutaneous tissue disorders		Pruritus	
Investigations		GFR decreased	Haemoglobin decreased

In the FIDELIO-DKD study, hyperkalaemia events were reported in 18.3% (516/2827) of finerenone-treated patients compared with 9.0% (255/2831) of placebo-treated patients. In patients treated with finerenone, the majority of hyperkalaemia events were mild to moderate and resolved. Serious events of hyperkalaemia were reported more frequently for finerenone (1.6%, 44/2827) than for placebo (0.4%, 12/2831). Serum potassium concentrations >5.5 mmol/l and >6.0 mmol/l were reported in 21.7% and 4.5% of finerenone treated patients and in 9.8% and 1.4% of placebo-treated patients, respectively.

Hyperkalaemia leading to permanent discontinuation in patients who received finerenone was 2.3% (64/2827) versus 0.9% (25/2831) in the placebo group. Hospitalisation due to hyperkalaemia in the finerenone group was 1.4% (40/2827) versus 0.3% (8/2831) in the placebo group. No fatal hyperkalaemia adverse events were reported.

Investigator reported treatment-emergent AEs related to hyperkalaemia



Adapted from Bakris[2]

*Investigator-reported AEs using the MedDRA preferred terms 'hyperkalemia' & 'blood potassium increased'

An increase from baseline in mean serum potassium was observed in the first month of finerenone treatment compared to placebo and a maximum between-group difference of 0.23 mmol/l at month 4. The difference in serum potassium between finerenone and placebo remained stable thereafter.

Other findings of note from FIDELIO-DKD[1,2]

Finerenone had modest effects on blood pressure: the changes in mean systolic blood pressure from baseline to month 1 and to month 12 were −3.0 and −2.1 mmHg, respectively, with finerenone and −0.1 and 0.9 mmHg, respectively, with placebo. Glycated haemoglobin levels were similar in the two groups.

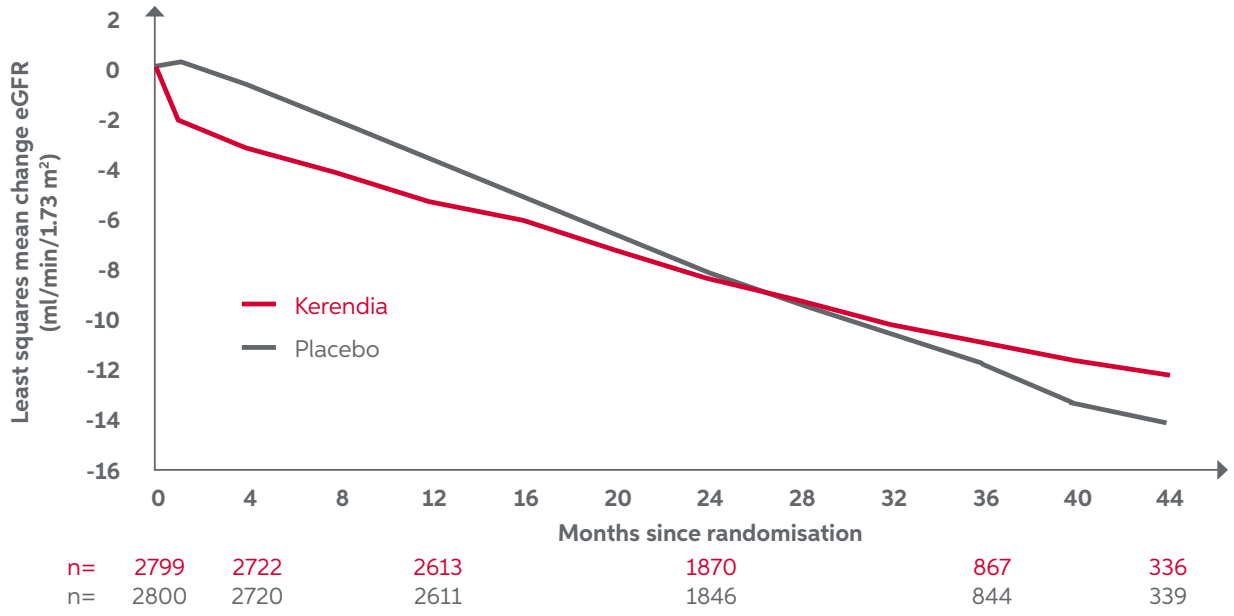
Sexual side effects, such as gynaecomastia, were rare and balanced between groups, and there were no incidences of breast hyperplasia reported with finerenone.

For further details regarding undesirable effects and recommendations regarding hyperkalaemia, see posology and special warnings and precautions for use above, and the summary of product characteristics.

Patients on finerenone experienced an initial decrease in eGFR (mean 2 ml/min/1.73 m^2) that attenuated over time compared to placebo. This decrease appeared to be reversible during continuous treatment.

Patients on Kerendia experienced an initial decrease in eGFR that attenuated over time vs placebo[2]

Mean change from baseline level in eGFR[2]



n=	2799	2722	2613	1870	867	336
n=	2800	2720	2611	1846	844	339

Adapted from Bakris[2]

Burden of disease

It is estimated that chronic kidney disease (CKD) is present in one in ten adults based on analysis of global data from 2022, but this varies according to how CKD is defined (possible/measured/diagnosed)[3]. Diabetic kidney disease (DKD) accounts for >40% cases of CKD globally.[4]

CKD in patients with type 2 diabetes (T2D) is a progressive disease associated with increased risk of kidney and cardiovascular (CV) complications and mortality.[5,6] People with diabetes and CKD have an earlier onset and greater severity of all complications associated with CKD in comparison with those without diabetes[7]. The presence of both CKD and T2D exacerbates CV risk, with an approximate 3 to 6-fold increase in the risk of CV mortality, compared to those patients with T2D and those with neither T2D nor kidney disease.[5]

CKD represents a significant burden on healthcare systems.[8] Indeed, in England, the cost of CKD was estimated at between £1.44 – £1.45 billion (2009-2010), around 1.3% of all NHS spending in that year.[9] Medical resource utilisation and associated costs increase as patients progress to more advanced CKD stages.[10]

The National Chronic Kidney Disease audit[11] found that for every 100 patients with chronic kidney disease and moderate to severe function impairment, there are 38 unplanned admissions per year and as kidney function worsens, there are more admissions per year; for every 100 patients with stage 4 CKD, there will be more than 70 unplanned admissions per year and for every 100 patients with stage 5 CKD, more than 110 unplanned admissions per year. Deaths also increase by stage, with the figures per 100 patient years being approximately 6, 19 and 25 in CKD stages 3-5 respectively.

68,111 adult patients were receiving renal replacement therapy (RRT) for ESKD in the UK on 31/12/2019 according to the UK renal registry. Diabetes is the most common identifiable primary renal disease (PRD) for patients starting RRT and its contribution has increased over recent years (23.7% in 2010 and 30.4% in 2019).[12]

Importantly, dialysis is an intervention that has a substantial impact on the life of patients and their family and/or caregivers. Indeed, some people with kidney failure will decide not to have dialysis treatment such is the burden it imposes. For those who are easily confused, for example, people who have dementia, dialysis may seem frightening or upsetting.[13] Several publications have reported a negative impact of dialysis on carers quality of life, with a particular impact on mental health.[14-16]

CKD definition and classification

CKD is defined as abnormalities of kidney function or structure present for more than 3 months, with implications for health. This includes all people with markers of kidney damage (such as proteinuria) and those with a glomerular filtration rate (GFR) of less than 60 ml/min/1.73 m^2 on at least 2 occasions separated by a period of at least 90 days (with or without markers of kidney damage).[17]

The NICE guideline on CKD[17], recommends that testing for CKD using eGFR creatinine and albumin-to-creatinine ratio (ACR) should be offered to adults with diabetes. It is important to classify CKD using GFR and ACR categories as increased ACR is associated with increased risk of adverse outcomes, decreased GFR is associated with increased risk of adverse outcomes and increased ACR and decreased GFR in combination multiply the risk of adverse outcomes. The National Chronic Kidney Disease audit found that on average GPs test 86% of people with diabetes for CKD (using annual blood tests), but only 54% have the relevant annual urine tests. The audit also found that 70% of biochemically confirmed cases of CKD were given an appropriate Read code.[11]

The most widely used CKD classification system was developed by KDIGO (Kidney Disease: Improving Global Outcomes) and is illustrated in the figure below, reproduced from.[18]

Prognosis of CKD by GFR and albuminuria category

GFR categories (ml/min/1.73 m^2) Description and range			Persistent albuminuria categories Description and range		
			A1	A2	A3
			Normal to mildly increased	Moderately increased	Severely increased
			<3 mg/mmol	3-30 mg/mmol	>30 mg/mmol
G1	Normal or 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			

low risk (if no other markers of kidney disease, no CKD)
moderately increased risk
high risk
very high risk

Adapted from KDIGO Diabetes Work Group[18]

Prevalence

The prevalence of GP recorded CKD with classification G3a to G5, for data up to March 2021, was 3.8%, with prevalence increasing with age to 28.9% in those aged 80+ years.[19]

A report from Public Health England in 2014[20] reported that it is expected that 2.6 million people aged 16 years and older are living with CKD stage 3-5 (diagnosed and undiagnosed). This is equal to 6.1% of the population of this age group. Considering the ageing population, this report also projected that prevalence of CKD stage 3-5 is expected to increase to 4.2 million people in 2036.

Diabetic kidney disease (DKD) accounts for >40% cases of CKD.[4]

Equality considerations

CKD may disproportionately affect patients from lower socio-economic groups and those from Black, Asian and minority Ethnic populations.

A report by Kidney Research UK[21] reported that people from lower socio-economic groups are more likely to (1) have risk factors associated with CKD such as diabetes and hypertension, (2) develop CKD, (3) progress faster towards kidney failure, (4) die earlier with CKD, (5) be diagnosed at a later stage of the disease, (6) have poorer survival rates on dialysis.

The same report stated that people from Black, Asian and Minority Ethnic populations (1) have a greater burden of risk factors for kidney disease such as diabetes and hypertension, (2) are more likely to progress faster towards kidney failure, (3) are less likely to receive a kidney transplant, (4) have a different pattern of

uptake of home dialysis therapies. In addition, people from South Asian and Black backgrounds are 3-5 times more likely to start dialysis than people from Caucasian backgrounds.

Existing management and place in therapy

Optimal treatment of CKD in T2D is facilitated by early detection, hence the importance of regular CKD screening in patients with diabetes. Identification of patients with early signs of CKD enables implementation of disease management strategies to reduce the risk of progression to end-stage renal disease (ESRD) and of CV events, thereby improving patient outcomes and reducing the impact of CKD on healthcare resources.[22,23]

NICE published an update to their guidelines in 2021 on CKD assessment and management.[17] NICE also updated their guidelines on type 2 diabetes in adults: management.[24] Management includes lifestyle and dietary advice as well as pharmacotherapy. Medication for patients with type 2 diabetes and CKD includes drugs to manage glycaemia, CV disease and ongoing risk as well as antihypertensives and drugs to manage persistent proteinuria. For many years, ACEis/ARBs have been the standard of care treatments for patients with CKD in T2D based on their evidence for retarding the progression toward end-stage renal disease.[25-28]

Recently updated NICE guidelines recommend SGLT2i[24] as follows:

- For adults with type 2 diabetes and CKD who are taking an ARB or an ACE inhibitor (titrated to the highest licensed dose that they can tolerate), offer an SGLT2 inhibitor (in addition to the ARB or ACE inhibitor) if:
  - ACR is over 30 mg/mmol and
  - they meet the criteria in the marketing authorisation (including relevant estimated glomerular filtration rate [eGFR] thresholds).
- For adults with type 2 diabetes and CKD who are taking an ARB or an ACE inhibitor (titrated to the highest licensed dose that they can tolerate), consider an SGLT2 inhibitor (in addition to the ARB or ACE inhibitor) if:
  - ACR is between 3 and 30 mg/mmol and
  - they meet the criteria in the marketing authorisation (including relevant eGFR thresholds).

SMC have issued their advice on dapagliflozin as follows:[29]

Dapagliflozin is accepted for restricted use within NHSScotland.

Indication under review: in adults for the treatment of chronic kidney disease.

SMC Restriction:

- in patients with an estimated glomerular filtration rate of ≥25 to ≤75 mL/min/1.73m² at treatment initiation, and
- are receiving an angiotensin converting enzyme inhibitor or angiotensin receptor blocker (unless these are not tolerated or contraindicated), and
- have a urine albumin creatinine ratio of at least 23mg/mmol, or type 2 diabetes mellitus or both.

In a randomised, double-blind, phase III study in patients with chronic kidney disease, treatment with dapagliflozin added to standard of care significantly reduced the risk of first occurrence of ≥50% sustained decline in estimated glomerular filtration rate, end stage renal disease, cardiovascular death or renal death when compared with standard of care alone.

Unmet need

The burden of disease is significant as described above.

Despite standard of care therapy and recent emerging therapies, overall, there remains a residual risk of progressive deterioration in kidney function in patients with CKD and T2D.[26,29-32] Hence, there is a need for additional treatment options to further reduce cardiorenal morbidity and mortality in patients with CKD and T2D.

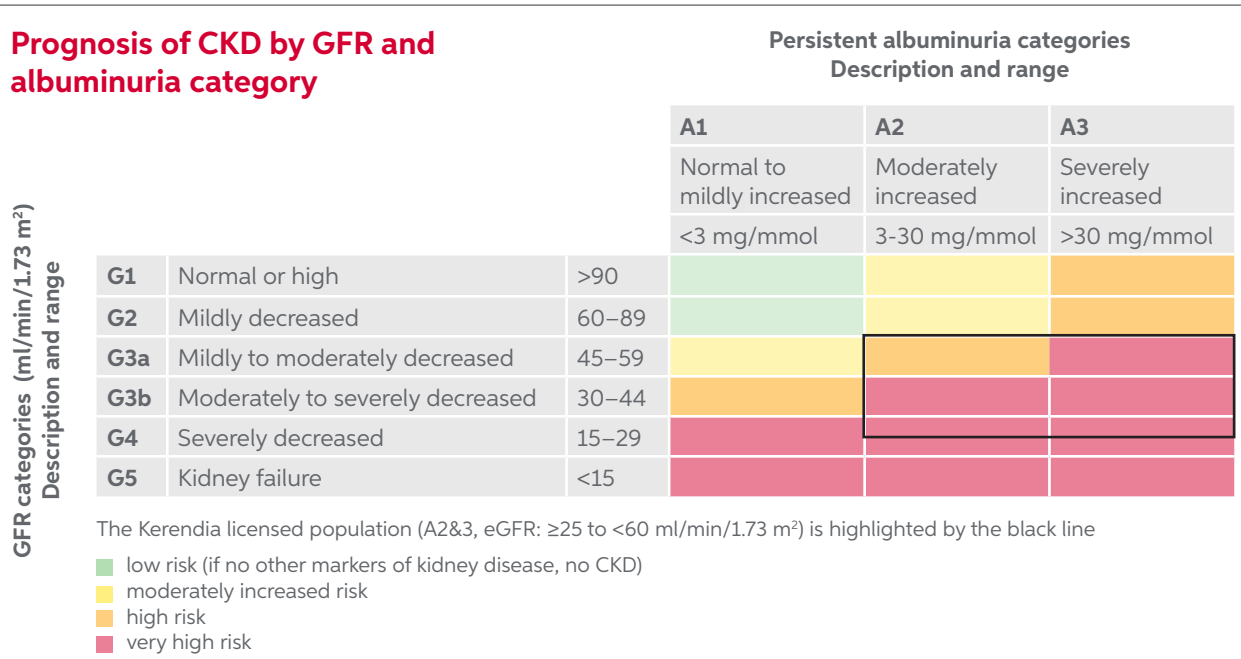
Patient numbers potentially eligible for finerenone

Finerenone is indicated for the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults.

In patients with an eGFR between ≥25 to <60 ml/min/1.73 m², finerenone may be initiated depending on their serum potassium. According to the licensed indication, finerenone can be initiated in patients with CKD stage 3 and 4. This is equivalent to eGFR ≥15 to <60 ml/min/1.73 m². However, in patients with eGFR <25 ml/min/1.73 m², finerenone treatment should not be initiated due to limited clinical data.[1]

This is illustrated on the KDIGO grid below.

Prognosis of CKD by GFR and albuminuria category



Adapted from KDIGO Diabetes Work Group[18]

An analysis of CPRD[32], has identified that 4.84% of patients with type 2 diabetes meet the criteria for initiation of finerenone. This source also found that 63.5% of patients with CKD and type 2 diabetes are taking ACEi/ARB, a prerequisite of the study protocol (although it is not known how many of these patients in CPRD are taking ACEi/ARB therapy at a maximum labeled dose that does not cause unacceptable side effects).

Using these figures and applying them to the Scottish population and diabetes prevalence data, gives an estimated population eligible for finerenone in 2023 of approximately 6,985.

Estimated eligible population	
	2023
Scottish population (aged 18+)[33]	4,467,751
Diabetes prevalence (2020)[34]	5.8%
Proportion with type 2 diabetes[34]	87.7%
CKD criteria for initiation of finerenone[32]	4.84%
Proportion of patients taking ACEi/ARB[32]	63.50%
Estimated eligible population for Scotland (with rounding)	6,985

Considering an adult population of 100,000, the estimated eligible population for finerenone is 156 patients (6,985/44.68).

NHS list price per month

28 tablets of 10 mg have an NHS list price of £36.68.

28 tablets of 20 mg have an NHS list price of £36.68.

Estimated budget impact per 100,000 adult population

Based on the estimated eligible population and NHS list price above, if all eligible patients were prescribed finerenone and stayed on treatment, the cost per year would be £74,387 [365 days/28 day pack ~13 packs/year].

Assessment of cost effectiveness

Finerenone has been shown in economic modelling by Bayer to be a cost-effective treatment for NHS Scotland with an Institute for Clinical Economic Review of £11,578 which was robust to various sensitivity and scenario analysis.

Relevant national / international guidelines

	Date	Recommendation
SMC[35]	2022	"Finerenone (Kerendia®) is accepted for use within NHSScotland. Indication under review: for the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults" "In a randomised, double-blind, phase III study, the addition of finerenone to angiotensin-converting enzyme inhibitor or angiotensin receptor blocker reduced the risk of the primary composite renal outcome comprising kidney failure, a sustained decrease in estimated glomerular filtration rate of ≥40% or death from renal causes compared with placebo"
NICE[36]		"1.1 Finerenone is recommended as an option for treating stage 3 and 4 chronic kidney disease (with albuminuria) associated with type 2 diabetes in adults. It is recommended only if: • it is an add-on to optimised standard care; this should include, unless they are unsuitable, the highest tolerated licensed doses of: o angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs) and o sodium–glucose cotransporter-2 (SGLT2) inhibitors and • the person has an estimated glomerular filtration rate (eGFR) of 25 ml/min/1.73 m^2 or more." "1.2 This recommendation is not intended to affect treatment with finerenone that was started in the NHS before this guidance was published. People having treatment outside this recommendation may continue without change to the funding arrangements in place for them before this guidance was published, until they and their NHS clinician consider it appropriate to stop."
UKKA – UK Kidney Association Clinical Practice Guideline: Sodium-Glucose Co-transporter-2 (SGLT-2) Inhibition in Adults with Kidney Disease	2021	"There is likely to be increasing use of MRA in CKD populations due to recent positive results from finerenone trials and guideline recommendations"
American Association of Clinical Endocrinology (AACE) guidelines (2022)[37]	2022	"A nonsteroidal mineralocorticoid receptor antagonist (finerenone) with proven kidney and CVD benefit is recommended for persons with T2D, an eGFR 25 mL/min/1.73 m^2, normal serum potassium concentration, and albuminuria (UACR 30 mg/g) despite a maximum tolerated dose of a renin-angiotensin system inhibitor" Grade A; best evidence level (BEL 1)
KDIGO	2022	• Recommendation 1.4.1: "We suggest a nonsteroidal mineralocorticoid receptor antagonist with proven kidney or cardiovascular benefit for patients with T2D, an eGFR ≥25 ml/min per 1.73 m^2, normal serum potassium concentration, and albuminuria (≥30 mg/g [≥3 mg/mmol]) despite maximum tolerated dose of RAS inhibitor (RASi) (2A)" • Practice Point 1.4.1: "Nonsteroidal MRA are most appropriate for patients with T2D who are at high risk of CKD progression and cardiovascular events, as demonstrated by persistent albuminuria despite other standard-of-care therapies" • Practice Point 1.4.2. "A nonsteroidal MRA can be added to a RASi and an SGLT2i for treatment of T2D and CKD" • Practice Point 1.4.3: "To mitigate risk of hyperkalemia, select patients with consistently normal serum potassium concentration and monitor serum potassium regularly after initiation of a nonsteroidal MRA"

ADA – Standards of Medical Care in Diabetes[38]	2022	"In patients with CKD who are at increased risk for CV events or CKD progression or are unable to use a SGLT2i, a nonsteroidal MRA (finerenone) is recommended to reduce CKD progression and CV events" "For patients with type 2 diabetes and CKD treated with maximum tolerated doses of ACE inhibitors or angiotensin receptor blockers, addition of finerenone should be considered to improve CV outcomes and reduce the risk of CKD progression" "Patients with type 2 diabetes and CKD should be considered for treatment with finerenone to reduce CV outcomes and the risk of CKD progression"
ADA/ KDIGO consensus[39]	2022	"A nonsteroidal mineralocorticoid receptor antagonist with proven kidney and cardiovascular benefit is recommended for patients with T2D, an eGFR ≥25 ml/min/1.73 m^2, normal serum potassium concentration, and albuminuria (ACR ≥30 mg/g) despite maximum tolerated dose of RAS inhibitor"

Example shared care template for local adoption

Proposed shared care arrangements for finerenone		
Stage of treatment	**Consultant or specialist in primary care**	**GP**
Initiation	Assess patient following referral Carry out baseline tests required prior to initiation of finerenone Initiate finerenone 10 mg od	n/a
Maintenance	Assess clinical response to treatment Titrate to target dose of 20 mg od where clinically indicated Provide advice to non-specialist as required	Continue treatment Monitor renal function & serum potassium as per SmPC

Reference pack

SmPC for Kerendia 10 mg & 20 mg tablets

FIDELIO-DKD paper & editorial

Selected reviews relevant to use of non-steroidal MRAs in patients with diabetic kidney disease

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For more information

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