PULSE

This quick guide has been funded by Bayer plc. through a reactive sponsorship. Bayer plc. had no involvement with the development of this educational guide.

O2O CREATIVE / E+ / VIA GETTY IMAGES

QUICK GUIDE

Chronic kidney disease in patients with type 2 diabetes: A guide for General Practitioners

Chronic kidney disease (CKD) is a significant complication in patients with type 2 diabetes (T2DM), affecting approximately 40% of those with T2DM in the UK.[1] CKD is defined as an abnormality in kidney function or structure (or both) present for more than 3 months.

General practitioners (GPs) play a critical role in the early identification, management and prevention of progression of CKD in patients with T2DM.

This article provides an overview for GPs, focusing on the pathophysiology, diagnosis and management of CKD in T2DM, in line with relevant UK guidelines.

Understanding CKD in T2DM

CKD in T2DM is characterised by a progressive decline in kidney function, often due to inflammation and scarring caused by prolonged hyperglycaemia. This results in impaired renal filtration, leading to protein leakage into urine (albuminuria) and a reduced estimated glomerular filtration rate (eGFR). CKD is staged

Continued overleaf →

CKD Stage	eGFR
Stage 1 (G1)	Normal (≥90ml/min/1.73m^2) but with markers of kidney damage
Stage 2 (G2)	Slightly reduced (60–89ml/min/1.73m^2) and with other markers of kidney damage
Stage 3a (G3a)	45–59ml/min/1.73m^2
Stage 3b (G3b)	30–44ml/min/1.73m^2
Stage 4 (G4)	15–29ml/min/1.73m^2
Stage 5 (G5)	<15ml/min/1.73m^2 (kidneys have lost almost all of their function)

Table 1. CKD Stages by eGFR[2,3]

from 1 to 5 based on eGFR and albuminuria levels, with stages 3–5 indicating significant kidney impairment.[2,3] See Table 1.

Diabetes-related CKD is a leading cause of end-stage kidney disease (ESKD), defined as eGFR <15ml/min/1.73m^2 or requiring dialysis or kidney transplant, in the UK, contributing to approximately one-third of cases requiring renal replacement therapy.[1] Patients with CKD and T2DM are also at high risk of macrovascular cardiovascular disease (CVD) events, including myocardial infarction (MI), heart failure and CVD death, so holistic management is essential.

Diagnosis and screening

GPs are often the first point of contact for diagnosing CKD in patients with T2DM. Early identification is critical, as CKD progresses silently, with symptoms often absent until advanced stages. NICE guidelines recommend at least annual screening for CKD in patients with T2DM using two key tests:[2]

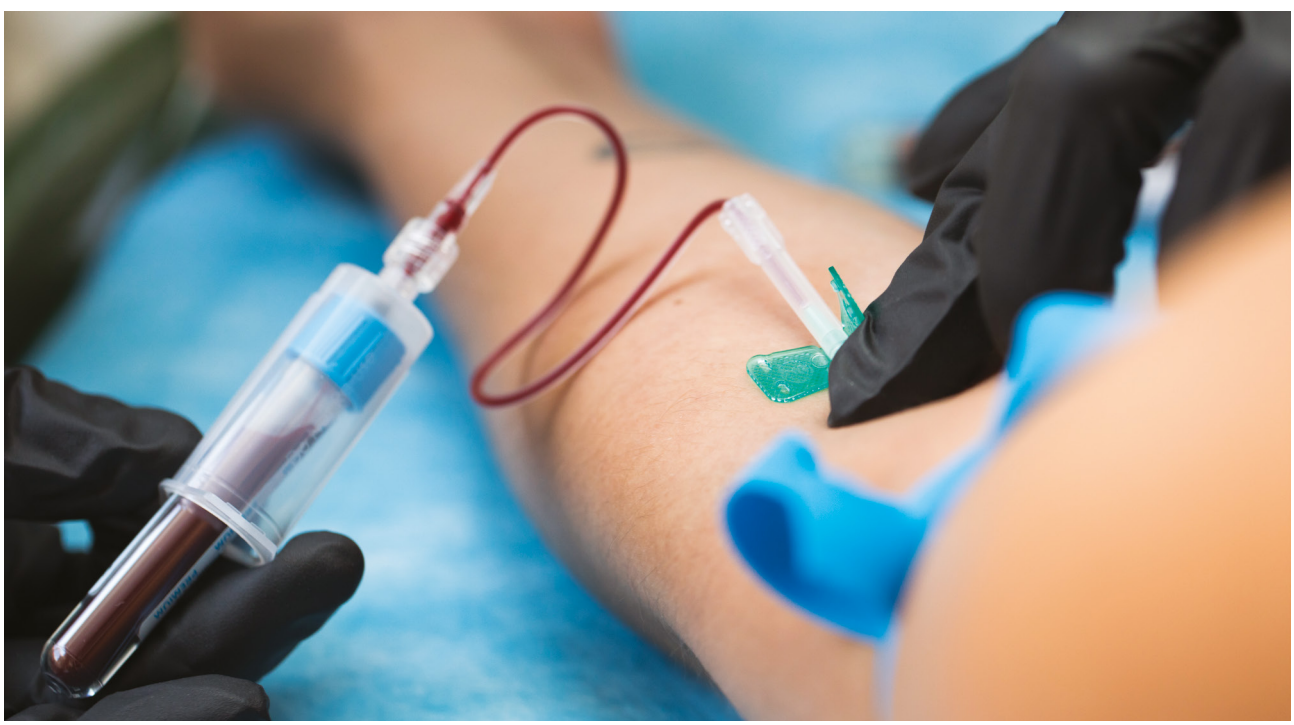
1 Urine Albumin-to-Creatinine Ratio (uACR): A uACR >3 mg/mmol indicates albuminuria, an early marker of kidney damage. Persistent albuminuria (>30mg/g or >3mg/mmol) is a key indicator for initiating specific therapies.

2 Estimated Glomerular Filtration Rate (eGFR): An eGFR <60 mL/min/1.73 m², confirmed by two measurements at least 90 days apart, defines CKD stages 3–5.

GPs should ensure these tests are performed regularly, as early detection enables interventions to slow CKD progression. Coding CKD in patient records is crucial, as it facilitates appropriate care plans and safe prescribing. Studies show that only 55–70% of patients with biochemical CKD are appropriately coded in UK primary care,[4] with factors like older age, male sex, diabetes, and hypertension associated with better coding rates. GPs should prioritise accurate coding to improve patient outcomes.

Management strategies

The management of CKD in T2DM focuses on: managing glycaemia, blood pressure and lifestyle factors; slowing kidney disease progression; and reducing CVD risk.[1,2,5-7]



OJOS DE HOJALATA/ VIA GETTY IMAGES

This quick guide has been funded by Bayer plc. through a reactive sponsorship. Bayer plc. had no involvement with the development of this educational guide.

1 Glycaemic control

Glycaemic control is essential to prevent microvascular complications in patients with T2DM. NICE recommends metformin (alongside lifestyle advice) as first-line therapy for patients with CKD and T2DM.[5] Note however that metformin is contraindicated in patients with eGFR <30ml/min/1.73m² and dose reductions are advised with more moderate renal impairment – see product literature for details.[8]

Sodium-glucose cotransporter-2 (SGLT-2) inhibitors should be added in many T2DM patients with CKD (and other T2DM patients with or at risk of CVD) as they have been shown to reduce CKD progression and CVD events (see points 3 and 4).[2,5]

Blood glucose targets should be tailored to the individual, aiming to achieve a glycated haemoglobin (HbA1c) level <48mmol/mol (6%) with a single drug not associated with hypoglycaemia, or HbA1c <53mmol/mol (7%) in those on a drug associated with hypoglycaemia.[5] Escalation of therapy with additional medication if needed to achieve blood glucose targets should follow the recommended treatment pathway in NICE T2DM guidance.

2 Blood pressure management

Hypertension is a major driver of CKD progression.[1] NICE recommends adults with CKD and T2DM diagnosed with hypertension should be treated with an ACE inhibitor or ARB first line, with the target blood pressure of <140/90mmHg in those with uACR <70mg/mmol, or <130/80mmHg with uACR ≥70mg/mmol (unless the patient is frail, in which case blood pressure targets should be relaxed according to clinical judgement).[6] GPs should follow the NICE stepwise treatment algorithm to escalate antihypertensive therapy as required.

3 Slowing kidney disease progression

NICE recommends that individuals with CKD and T2DM and persistent proteinuria (uACR ≥3mg/mmol) should be offered an ACE inhibitor or ARB irrespective of whether diagnosed with hypertension.[2]

SGLT-2 inhibitors are also now recommended as an add-on therapy to an ACE inhibitor or ARB for CKD associated with T2DM, to help slow disease progression.[2]

Specifically, for people with T2DM and CKD who are on an ACE inhibitor or ARB (titrated to the highest tolerated dose), NICE recommends GPs should:

- offer an SGLT-2 inhibitor if uACR is >30mg/mmol
- consider offering an SGLT-2 inhibitor if uACR is 3-30 mg/mmol

provided eGFR thresholds for the specific agent are met (dapagliflozin, eGFR 25–75ml/min/1.73m²; empagliflozin, eGFR 45-90ml/min/1.73m²).[2]

NICE now also recommends the non-steroidal mineralcorticoid antagonist (MRA) finerenone as an option in patients with stage 3 or 4 CKD (with albuminuria) associated with T2DM.[9] Finerenone has been shown to reduce progression of CKD and CVD events.[1]

NICE advises finerenone as an option for stage 3 or 4 CKD (with albuminuria, specifically: uACR persistently ≥3mg/mmol [≥30mg/g]) associated with T2DM in adults, where:

- It is an add-on to optimised standard care (including highest tolerated licensed doses of ACE inhibitors or ARBs and SGLT-2 inhibitors).
- The person has an eGFR of ≥25ml/min/1.73m to <60ml/min/1.73m.[9]

4 CVD risk reduction

NICE recommends low-dose aspirin for secondary prevention in those with established CVD.[7]

SGLT-2 inhibitors also reduce CVD events, and are now a cornerstone of therapy in patients with CKD and T2DM (see points 1 and 3).[2,5]

NICE recommends patients with CKD and T2DM should routinely be prescribed lipid-lowering therapy for primary and secondary CVD prevention, starting with atorvastatin at 20mg for primary and 80mg for secondary prevention.[7]

5 Lifestyle and dietary interventions

Lifestyle modification is critical. GPs should advise patients on smoking cessation, weight management and regular physical activity to reduce cardiovascular and kidney risks. Referral to dietitians or diabetes educators can support adherence to these recommendations.

Challenges in primary care

GPs face barriers in managing CKD in T2DM, including undercoding of CKD, which limits access to guideline-directed therapies. Time constraints, patient complexity, and varying adherence to screening protocols can further complicate care. GPs should use practice-level audits to enhance CKD coding and management.

Key points

- GPs in England are pivotal in managing CKD in patients with T2DM.
- Regular screening with uACR and eGFR, accurate coding and adherence to NICE guidelines are essential for early intervention.
- Therapies including SGLT-2 inhibitors, ACE inhibitors/ARBs and non-steroidal MRA (for stages 3–4 CKD with albuminuria) can significantly slow CKD progression and reduce CVD risk.
- By integrating guideline-directed medical therapy with lifestyle interventions, GPs can improve outcomes for this high-risk population.

References

1 Dasgupta I et al. Current management of chronic kidney disease in type-2 diabetes—A tiered approach: An overview of the joint Association of British Clinical Diabetologists and UK Kidney Association (ABCD-UKKA) guidelines. *Diabet Med* 2025 Feb;42(2):e15450

2 NICE. Chronic Kidney Disease: Assessment and Management. [NG203] 2021

3 NHS UK. Conditions. Chronic kidney disease: Diagnosis. Available at: https://www.nhs.uk/conditions/kidney-disease/diagnosis/

4 Kim L et al. How do primary care doctors in England and Wales code and manage people with chronic kidney disease? Results from the National Chronic Kidney Disease Audit. *Nephrol Dial Transplant* 2018;33(8):1373-79

5 NICE. Type 2 Diabetes in Adults: Management. [NG28] 2022

6 NICE. Hypertension in adults: diagnosis and management. [NG136] 2023

7 NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification. [NG238] 2023

8 British National Formulary. Metformin hydrocholoride: Renal impairment. Available at: https://bnf.nice.org.uk/drugs/metformin-hydrochloride

9 NICE. Finerenone for treating CKD in T2DM. [TA877] 2023

Kerendia® finerenone

This promotional material has been developed and funded by Bayer plc and is intended for UK healthcare professionals only. Kerendia (finerenone) is indicated for the treatment of chronic kidney disease (stage 3 and 4 with albuminuria) associated with type 2 diabetes in adults.
Prescribing information and adverse event reporting information can be found here or accessed via a QR code located at the bottom of this document.

BAYER

Kerendia® a non-steroidal MRA, is distinct from steroidal MRAs

The mineralocorticoid receptor antagonists differ in their licensed indications:[1-3]

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

Spironolactone[2]
Indicated in nephrotic syndrome.

Eplerenone[3]
No licensed renal indication.

Pharmacological differences between Kerendia and steroidal MRAs[4,5]

Based on preclinical data, not supported by human studies. Kerendia® has not been compared to currently available MRAs in phase 3 clinical trials. The clinical consequences of differences between the characteristics described is therefore unknown.

CHARACTERISTIC	KERENDIA®	SPIRONOLACTONE	EPLERENONE
MRA class[4]	Non-steroidal	Steroidal	Steroidal
Selectivity for MR[4]	High	Low	Medium
Potency for MR[4]	High	High	Low
Metabolites[4]	No active metabolites	Multiple active metabolites	No active metabolites
BP-lowering effect[5]	Weak*	Strong	Weak

*The majority of hypotension events were mild or moderate and resolved in patients treated with Kerendia®. For further information consult the Kerendia® Summary of Product Characteristics.[1]

Kerendia® has no relevant affinity for glucocorticoid, androgen, oestrogen or progesterone receptors[6]

In keeping with its non-steroidal structure, hormonal adverse events (reproductive system and breast disorders) with Kerendia® were similar to placebo; 126 (4.5%) vs. 146 (5.2%) respectively.[7]

In the pivotal phase III FIDELIO-DKD study, gynaecomastia occurred in 6 (0.2%) of patients in both the Kerendia® and placebo arms.[7] Kerendia® has been shown to have an anti-fibrotic and anti-inflammatory effect in animal models[8,9]

The most frequently reported adverse reaction under treatment with Kerendia® was hyperkalaemia[1]

KERENDIA IS THE ONLY MRA RECOMMENDED FOR CKD (STAGE 3 AND 4 WITH ALBUMINURIA):

NICE recommended[10]

SMC accepted[11]

- The CVRM diseases, CVD, CKD and T2D are closely interlinked and the interaction between these conditions requires a holistic approach to care.[12]
- Availability of a therapy like Kerendia® with cross-indications across CKD and T2D[1] is therefore of paramount importance.

Protect the kidneys, support the heart

Explore the connection between CKD and CVD here:

NICE recommends Kerendia®, the first and only UK licensed non-steroidal MRA, as an add-on to standard of care for stage 3 and 4 CKD (with albuminuria) associated with T2D[10]

Kerendia® slows CKD progression in T2D and can significantly delay progression of renal disease (vs. placebo)[13]

Diabetic kidney disease is progressive and irreversible; act now with Kerendia® to significantly reduce the risk of renal & CV events for your patients (vs. placebo)[13]

CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; CVRM, cardiovascular-renal-metabolic; MR, mineralocorticoid receptor; MRAs, mineralocorticoid receptor antagonists; NICE, National Institute for Health and Care Excellence; SMC, Scottish Medicines Consortium; T2D, type 2 diabetes; UK, United Kingdom.

1. Kerendia SmPC; 2. Spironolactone SmPC; 3. Eplerenone SmPC; 4. Adapted from Kolkhof P, et al. Handb Exp Pharmacol 2017; 243:271–305; 5. Kobayashi M, et al. Nephrol Dial Transplant 2024;39:1063–1072; 6. Frampton JE. Drugs 2021;81(15):1787–1794; 7. Bakris GL, et al. N Engl J Med 2020;383:2219–2229 (supplementary appendix); 8. Kolkhof P, et al. J Cardiovasc Pharmacol 2014;64:69–78; 9. Grune J, et al. Hypertension 2018;71:599–608; 10. NICE. Finerenone for treating chronic kidney disease in type 2 diabetes [TA877]. Available at: https://www.nice.org.uk/guidance/ta877. Accessed March 2025; 11. SMC. finerenone (Kerendia). Available at: https://scottishmedicines.org.uk/medicines-advice/finerenone-kerendia-full-smc2486/. Accessed April 2025; 12. Kadowaki T, et al. Diabetes Obes Metab 2022;24(12):2283–2296; 13. Bakris GL, et al. N Engl J Med 2020;383:2219–2229.

Prescribing information for Kerendia® (finerenone) is available via the QR code on the right. Either click here or scan the QR code for prescribing information and adverse event reporting information. For direct access to this prescribing information, please ensure that your device's browser settings have automatic PDF download enabled.

Code PP-KER-GB-0818 **Date of preparation** June 2025