# ABCD & UKKA 2025

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## What's new in the management of diabetic kidney disease

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**Abstract**

Diabetic kidney disease (DKD) management is a rapidly evolving field. Alongside risk prediction tools, such as the Kidney Failure Risk Equation, there is greater emphasis on person-centred care and education.

For people with type 2 diabetes (T2DM), sodium-glucose cotransporter 2 inhibitors (SGLTi) and finerenone have joined the five previous pillars of care which are foundations for both type 1 diabetes (T1DM) and T2DM. These include blood glucose optimisation, blood pressure management, renin–angiotensin–aldosterone system inhibitors (RAASi), lipid management and smoking cessation. The evolution of hyperkalaemia management, due to the availability of potassium binders, has enabled the optimal use of RAASi.

Ongoing studies in people with T1DM will further inform DKD management in the future.

**Key words**: diabetic kidney disease, diabetic nephropathy, chronic kidney disease

### Introduction

Diabetic kidney disease (DKD) is common. Depending on the population studied, it affects between 20 to 40% of people with diabetes[1,2] Appropriate management of DKD reduces progression to end-stage kidney disease (ESKD) and morbidity and mortality due to cardiovascular disease.

People with DKD often fall between services and consequently miss out on standards of care. An observational population study in Sweden in 2024 demonstrated widespread therapeutic inertia and underuse of statins, renin–angiotensin–aldosterone system inhibitors (RAASi) and sodium-glucose cotransporter 2 inhibitors (SGLT2i) in people with chronic kidney disease (CKD).[3] This study also noted a delay between CKD detected using laboratory measurements and a recorded healthcare diagnosis, indicating that a significant number of people with CKD were undiagnosed and untreated.[3]

This finding will be familiar to healthcare practitioners. Discussion of kidney disease can cause surprise or alarm, despite longstanding evidence of renal impairment. People who are unaware of their diagnoses are unable to engage fully with management strategies. It is therefore unsurprising that education and person-centred care are embedded in recent CKD guidelines.[4] The introduction to the KDIGO 2024 clinical practice guideline for the evaluation and management of CKD has the following patient foreword:

> *"In an ever-increasingly busy world of medical care, as patients, we believe that the best approach is for any physician to aim to achieve a partnership of knowledge with the patient regarding their CKD care."*

Working alongside the person with DKD, after confirming a diagnosis, management falls into two main categories: first, monitoring and risk assessment; and second, treatment of risk factors and interventions to delay progression.

### Monitoring and risk assessment

NICE have clear guidelines for the frequency of monitoring of estimated glomerular filtration rate (eGFR) and urine albumin creatinine ratio (ACR) depending on the CKD stage (Table 1).[5]

In addition, in recent years, the Kidney Failure Risk Equation (KFRE) has gained widespread recognition and popularity (https://kidneyfailurerisk.co.uk/). This can be shared and viewed together with the person with DKD. The short videos (1-2 minutes long) contain concise and informative summaries that can reinforce clinic discussions. Depending on the situation, a 5-year risk of kidney failure > 5% can also be used to prompt a referral to the renal team.

### Sick day rules

As part of monitoring and risk assessment, sick day rules guidance should be discussed and information sheets shared. If these are not available locally, there are nationally available resources, for example from Kidney Care UK, UKKA and Diabetes UK.

When discussing sick day rules, it should be emphasised that medications such as angiotensin-converting enzyme inhibitors (ACEi) are stopped when people are unwell but they should be restarted following recovery.

### Interventions to delay progression

Healthcare practitioners may be aware of the five-finger rule to delay progression of CKD:[6]

1. Blood glucose optimisation
2. Blood pressure management
3. RAASi
4. Lipid management
5. Smoking cessation

Use of the five-finger rule for people with DKD is invaluable in busy clinics, particularly when people have been attending for years and when previous management is not reviewed. Audits, cohort studies and population studies have consistently demonstrated therapeutic inertia with regard to the pillars of care of DKD management.

The ABCD-UKKA joint committee have written extended guidance for the management of hyperglycaemia,[7] blood pressure management,[8] and lipid management.[9] There is also available a concise summary of all the guidelines.[10]

**Table 1.** Suggested frequency of monitoring in CKD (adapted from NICE)[5]

| CKD stage | A1 (< 3 mg/mmol) | A2 (3 to 30 mg/mmol) | A3 (> 30 mg/mmol) |
|---|---|---|---|
| G1-2 (>60 ml/min) | ≥12 months | 12 months | ≤12 months |
| G3a (45 to 59 ml/min) | 12 months | 12 months | 6 months |
| G3b (30 to 44 ml/min) | 6-12 months | 6 months | ≤6 months |
| G4 (15 to 29 ml/min) | 6 months | 6 months | 4 months |
| G5 (<15 ml/min) | 3 months | 3 months | ≤3 months |

## Blood glucose optimisation

For people with T2DM, selection of medications with additional cardiorenal benefit should be prioritized. SGLT2i are discussed below. The emerging role of glucagon-like peptide-1 (GLP-1) receptor agonists in CKD is reviewed separately within this journal.

## Blood pressure management

Hypertension management reduces both DKD and cardiovascular disease progression. Treatment targets for blood pressure (BP) control are dependent on CKD stage as well as age and frailty.

For people with lower stages of CKD without albuminuria, the target BP is set higher at ≤140/90 mmHg. Where albuminuria is present, the target is lower at ≤130/80 mmHg. Intensive treatment is not recommended below systolic BP <120 mmHg due to the increased risks of falls and acute kidney injury.[8]

RAASi are indicated as first-line therapy (based on the benefits beyond BP reduction) with the addition of second-line agents, including diuretics, if targets are not met. Based on the STOP ACE trial, RAASi are recommended in people with eGFR <30 ml/min.

## Lipid management

There are marked differences nationally and internationally with regard to best practice for lipid management in DKD. This can cause confusion and may possibly explain why lipid management is often neglected.

The ABCD-UKKA lipid guidelines are divided into sections of monitoring, indications to start and stop treatment, and what to use.[9] People with T1DM and young-onset T2DM are rarely included in large lipid trials. Thus, where evidence is lacking, general population data have been extrapolated and CKD used as a CVD risk equivalent.

Treatment is advised in people with diabetes (type 1 and 2), CKD G1-2 with albuminuria A2-3 and, if aged under 30 years, with an additional cardiovascular risk factor. In CKD stage 3-5, treatment is advised regardless of additional risk factors. Atorvastatin 20 mg is the starting point for treatment, with uptitration and addition of agents such as ezetimibe where targets are not met.

For simplicity, the targets of total cholesterol <4 mmol/L, LDL cholesterol <1.8 mmol/L and non-HDL cholesterol <2.5 mmol/L were set across the board. A percentage reduction in baseline levels was felt to be impractical, as was a graded approach to therapy.

Beyond the five-finger rule for DKD, improvements in hyperkalaemia management and the use of SGLT2i and finerenone have been established as additional pillars of care.

## Hyperkalaemia management

Hyperkalaemia can restrict the use of RAASi. The acute management of hyperkalaemia is well known; however, the management of chronic hyperkalaemia is less familiar.[11] This is divided into the management of correctable factors, dietary advice and adjuncts.

The management of correctable factors includes optimizing glucose and bicarbonate, consideration of SGLT2i and loop diuretics, and treating constipation (avoiding potassium-containing laxatives such as macrogol and preferentially using agents such as lactulose).

Dietary advice has changed over the past few years with regard to potassium. In the past, renal dieticians recommended the avoidance of fruits and vegetables noted to be high in potassium. However, it has been observed that these contain high amounts of fibre, meaning that the potassium enters and exits the body. Currently, there is a focus on avoiding processed foods, which can contribute to constipation and where the potassium is not excreted.

Finally, potassium binders, lokelma (sodium zirconium cyclosilicate) and patiromer (patiromer sorbitex calcium), are available. These can be used to manage persistent hyperkalaemia, $K^+$ ≥6 mmol/L, in people with CKD stage 3b to 5 (not on dialysis) or people with heart failure receiving suboptimal RAASi. These agents facilitate the use of RAASi. When the RAASi is discontinued, the potassium binder is also discontinued.

## SGLT2i

SGLT2i reduce the progression of both CKD and heart failure (preserved and reduced ejection fraction). This makes them very attractive for people with DKD.

The kidney outcome trials studied different populations (Table 2). However, in general it is considered unnecessary to use specific SGLT2i based on these.

**Table 2.** Concise summary of SGLT2i kidney outcome trials

| CREDENCE | DAPA-CKD | EMPA-Kidney |
|---|---|---|
| 4,400 participants | 4,300 participants | 6,609 participants |
| All with type 2 diabetes | With and without diabetes | With and without DM (included type 1 DM) |
| All on RAASi | Around 98% on RAASi | Around 85% on RAASi |
| eGFR 30-90 ml/min and urine ACR 30-500 mg/mmol | eGFR 25-75 ml/min and urine ACR 20-500 mg/mmol | eGFR 20 to 45 ml/min OR eGFR 40-90 ml/min and urine ACR 20 mg/mmol |
| All with albuminuria | All with albuminuria | With and without albuminuria |
| 30% RRR in renal outcome | 40% RRR in renal outcome | 28% RRR in renal outcome |

Instead, the beneficial effects of SGLT2i may be viewed as a class effect.

SGLT2i can be initiated, in people with T2DM, at an eGFR above 15 to 30 ml/min (currently, dapagliflozin >15 ml/min, empagliflozin >20 ml/min and canagliflozin >30 ml/min).

In the kidney outcome trials, SGLT2i were continued until dialysis (with adequate preserved urine production). Thus, there is evidence for use in people with eGFR <20 ml/min. There is no evidence that the beneficial effects attenuate at eGFR <20 ml/min. In 2023, the FDA removed dialysis dependency from the list of contra-indications. However, there is currently insufficient evidence to make recommendations on the use of SGL2Ti in people receiving maintenance haemodialysis.

When the eGFR is <30 ml/min, SGLT2i do not affect glucose excretion so diabetes medications do not need to be adjusted. However, if the eGFR is >30 ml/min, then medications with hypoglycaemic potential, such as insulin and gliclazide, should be reduced by 20-50%.

The benefits of SGLT2i largely outweigh the risks of mycotic genital infections and diabetic ketoacidosis. However, people should be counselled appropriately and ideally given written information or website links with regard to side effects and sick day rules.

Regarding SGLT2i and the risk of lower limb amputation, this was only observed in the CANVAS trial. It was not observed in CREDENCE. However, based on sick day rules guidance, it is advisable to stop SGLT2i in people who are acutely unwell with an infected diabetic foot ulcer. In people who are not unwell, with chronic diabetic foot ulcers and DKD, the benefits of SGLT2i likely outweigh the risks.

Controversy exists regarding the use of SGLT2i in people with T1DM. Dapagliflozin initially had a licence (2019 to 2021) in people with T1DM and a BMI >27 kg/m$^2$ where insulin therapy was insufficient to provide adequate control. Currently, SGLT2i are not licensed for people with T1DM.

The EASE trials (Empagliflozin as Adjunctive to inSulin thErapy) looked at empagliflozin in people with T1DM. All participants received blood glucose and ketones meters and were educated in DKA sick day rules. Despite this, the incidence of DKA was higher in the empagliflozin-treated groups compared to placebo. 4.3% of participants on 10 mg empagliflozin had DKA events, compared with 3.3% in the 25 mg empagliflozin group and 1.2% of participants in the placebo group. Participants on 25 mg empagliflozin had more severe cases, with one fatal case due to delayed DKA diagnosis and treatment.[12]

Similarly, the DEPICT trials (Dapagliflozin Evaluation in Patients with Inadequately Controlled Type 1 Diabetes) showed an increased risk of DKA (4.1 % of participants on 5 mg dapagliflozin and 3.7% of participants on 10 mg) compared with placebo (0.4% of participants).[13]

Post-hoc analysis of the DEPICT trials looking at urine ACR demonstrated improvement compared to placebo over 52 weeks. There was a 13.3% reduction in urine ACR (95% CI -37.2 to 19.8) for dapagliflozin 5 mg and a 31.1% reduction for dapagliflozin 10 mg (-49.9 to -5.2).[14] There have been additional post-hoc analyses using prediction models, suggesting improved cardiovascular and renal risk. However, there has been a lack of dedicated renal outcome trials for T1DM.

There is a clear unmet need for adjunct therapy for people with DKD and T1DM, with the FinnDiane Study highlighting the increased mortality associated with progressive albuminuria.[15] Despite this, there is currently a lack of trial data clearly demonstrating efficacy of SGLT2i in T1DM. This, combined with the increased risk of DKA, indicates the requirement for further trials combined with careful patient selection and education.

## Finerenone

Finerenone is a third generation mineralocorticoid receptor inverse agonist with reduced side effects (hyperkalaemia and gynaecomastia) and preserved benefits compared to spironolactone.

Two phase 3, randomized, placebo-controlled, double-blind, multicentre studies looked at the safety and efficacy of finerenone, FIDELIO-DKD (Finerenone in reducing kidney failure and disease progression in DKD) and FIGARO-DKD (Finerenone in reducing cardiovascular mortality and morbidity in DKD). These complementary studies were analysed together in FIDELITY and combine data on more than 13,000 people with CKD (stage 1-4, eGFR >25 ml/min/1.73m$^2$). In FIDELITY, there was a significant relative risk reduction in the cardiovascular composite endpoint of 14% and in the kidney composite endpoint of 23%.[16]

Currently, finerenone is licensed for people with T2DM with an eGFR between 25 and 60 ml/min, albuminuria (urine ACR >3 mg/mmol), normokalaemia ($K^+$ $\leq$5 mmol/L) and on maximally tolerated RAASi and SGLT2i. For people who fulfil these criteria, initiation of 10 mg finerenone should be followed by careful monitoring for hyperkalaemia at four weeks. The dose can be increased to 20mg finerenone daily if the $K^+$ is $\leq$4.8 mmol/L and the dose should remain at 10 mg if the $K^+$ is 4.8 to 5.5 mmol/L. Finerenone should be discontinued if the $K^+$ is $\leq$5.5 mmol/L. It can be restarted at 10 mg once daily if the $K^+$ reduces $\leq$5.0 mmol/L. Following this, monitoring is in keeping with CKD stage and serum potassium levels. Currently, finerenone should be stopped when the eGFR drops below 15 ml/min.

For people with T1DM, as with SGLT2i, finerenone is not currently licensed. FINE-ONE, a randomised, prospective, double-blind study in people with T1DM and CKD, will look at whether finerenone influences urine ACR.[17]

## Conclusions

Whilst the most recent advances for people with T2DM and DKD have not yet migrated across to people with T1DM, it is worth remembering that combined risk factor modification (glycaemia, blood pressure and lipid management, RAASi and smoking cessation) is effective in reducing progression of DKD. In addition, with regard to RAASi, hyperkalaemia management has evolved. For people with T2DM, the additional pillars of care of SGLT2i and finerenone have proven benefit for both progression of DKD and reduction in cardiac outcomes.

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