Managing diabetes in the presence of renal impairment

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Chronic kidney disease is common in patients with diabetes and is associated with increased morbidity and mortality. This article discusses the challenges of maintaining good glycaemic control and addressing modifiable risk factors in patients with diabetes and renal impairment.

There are approximately four million people with diagnosed diabetes and around 550,000 who have undiagnosed diabetes in the UK. Diabetes is the commonest cause of chronic kidney disease (CKD) and end-stage renal disease, with renal impairment affecting around a third of UK patients with diabetes. The earliest clinical marker of diabetic nephropathy is microalbuminuria, and the UK Prospective Diabetes Study (UKPDS) estimated that, 10 years after a diagnosis of type 2 diabetes, a quarter of patients will develop microalbuminuria or macroalbuminuria.

The presence of CKD in patients with diabetes confers a significantly increased morbidity and mortality. The one-year mortality in people on dialysis with diabetes is one and a half times higher than for people on dialysis without diabetes.

Impaired renal excretory function presents challenges to prescribing for optimising glycaemic control and risk factor modification. The aim of this article is to enable clinicians to recognise modifiable risk factors of diabetic nephropathy and to optimise glycaemic control in the context of CKD.

Diagnosis of diabetic nephropathy

CKD is defined as abnormalities of kidney structure or function, present for greater than three months, with implications for health. Diabetic nephropathy describes several disease processes caused by diabetes and hyperglycaemia, which result in structural and functional abnormalities of the kidney. The grading and staging of diabetic nephropathy is illustrated in Table 1. A combination of hyperglycaemia-induced oxidative stress, inflammation and renal haemodynamic changes leads to glomerular hyperfiltration, initially causing an increased glomerular filtration rate (GFR). This progresses to cause mesangial thickening and interstitial fibrosis, which leads to loss of glomerular filtration.

The first clinical sign of diabetic nephropathy is the presence of microalbuminuria, which is best detected by screening...
for albumin/creatinine ratio (ACR) on a first-voided early morning urine sample. ACR 2.5–30mg/mmol in men or 3.5–30mg/mmol in women is abnormal and should ideally be repeated twice more. Elevated ACR in two out of three samples suggests the presence of microalbuminuria. Macroalbuminuria is a persistent ACR >30mg/mmol, and this degree of albuminuria is detectable on urine dipstick testing.

Risk factor modification

The aim of management of diabetic nephropathy is to slow renal disease progression, reduce the risk of cardiovascular disease and screen carefully for other microvascular complications of diabetes.

Lifestyle modification

Current smoking is a dose-dependent, independent risk factor for the progression of diabetic nephropathy. The risk of the progression of renal disease in ex-smokers compared to never-smokers is similar, thus all patients with diabetes who smoke should be advised to stop. High protein and high salt intake should be avoided. Physical activity, and weight loss in the overweight or obese, is associated with a positive effect on risk and progression of diabetic nephropathy.

Hypertension

Regular monitoring and management of blood pressure is essential for patients who have diabetes, especially if they have renal impairment. The blood pressure target is below 140/80mmHg, or below 130/80mmHg if there is evidence of micro- or macrovascular disease. An ACE inhibitor is the recommended first-line treatment for most patients.

If ACE inhibitors are not tolerated, angiotensin II-receptor blockers (ARBs) should be used, not only for their anti-hypertensive effect but also their independent renoprotective effects. In patients with diabetes, ACE inhibitors and ARBs have been shown to reduce albuminuria, progression to overt proteinuria, the rate of decline in renal function and the risk of cardiovascular morbidity and mortality. Reduction or elimination of albuminuria is regarded by some as a goal of therapy, since this is an independent predictor of decline in GFR.

Table 1. The prognosis of chronic kidney disease (CKD) using estimated glomerular filtration rate (eGFR) and albuminuria. Using albuminuria and GFR classification, patients can be stratified according to low, moderate, high or very high risk of progression to end-stage renal disease. From: NICE. Chronic Kidney Disease in Adults: Assessment and Management. July 2014

<table>
<thead>
<tr>
<th>Persistent albuminuria categories, description and range</th>
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<tr>
<td></td>
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<tr>
<td>Normally to mildly increased</td>
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<td>&lt;30mg/g &lt;3mg/mmol</td>
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<table>
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<tr>
<th>GFR categories (ml/min/1.73m²), description and range</th>
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<tr>
<td>G1</td>
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<td>G2</td>
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<td>G3a</td>
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<td>G3b</td>
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<td>G4</td>
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<td>G5</td>
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Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red: very high risk

Table 1. The prognosis of chronic kidney disease (CKD) using estimated glomerular filtration rate (eGFR) and albuminuria. Using albuminuria and GFR classification, patients can be stratified according to low, moderate, high or very high risk of progression to end-stage renal disease. From: NICE. Chronic Kidney Disease in Adults: Assessment and Management. July 2014.
Glucose management

The 2015 NICE pathway for glucose management in patients with type 2 diabetes is shown in Figure 1, which is also applicable to the management of glucose in patients with diabetes and CKD.

Glycaemic targets

Tight glycaemic control from the time of diabetes diagnosis reduces the risk of developing diabetic nephropathy. Although improving glycaemic control is important to reduce other microvascular complications, there is no clear evidence that it will slow the progression of established CKD.

Glycaemic targets, therefore, should be tailored to the individual, balancing risk of hypoglycaemia with potential improvement in microvascular complications. People who have diabetes and significant CKD (eGFR <30ml/min/1.73m²), and especially those on dialysis, are at high risk of hypoglycaemia. Therefore, a modest glycaemic target may be sought, perhaps a glycated haemoglobin (HbA₁c) between 53 and 68mmol/mol. Tight glycaemic control for patients with severe renal failure or those on dialysis confers an increased risk of mortality, without a survival benefit.

Monitoring of glycaemia

NICE guidelines recommend monitoring HbA₁c at three to six monthly intervals until stable, then six monthly thereafter. It should be noted, however, that in patients with significant renal dysfunction, the HbA₁c may not be accurate due to abnormal red cell turnover, uraemia, blood transfusions, iron deficiency and metabolic acidosis. Therefore, regular capillary blood glucose monitoring (CBGM) should be encouraged, especially in patients on insulin or sulfonylureas.

Oral hypoglycaemic agents

Table 2 summarises the renal cut-off values for prescribing oral antihyperglycaemic agents for patients with renal impairment.

Metformin

Metformin, the first-line oral agent for treating type 2 diabetes, improves insulin sensitivity by decreasing hepatic glucose output and increasing peripheral glucose uptake. Its efficacy is equal to sulfonylureas, without the risk of hypoglycaemia. The maximum efficacious dosage is 2000mg daily. A 35% treatment failure rate has been reported for metformin monotherapy and 5% of people treated cannot tolerate it, even at a reduced dosage.

Metformin dose reduction should be considered in patients with eGFR less than 40ml/min/1.73m² and stopped at an eGFR less than 30ml/min/1.73m². The prescription and renal function should be reviewed frequently in the context of factors that could cause an acute kidney injury such as sepsis or dehydration and in some cases, metformin should be stopped until kidney function has returned to baseline. Metformin-associated lactic acidosis is reported, but appears to be extremely rare (3 per 100,000 patient-years) and has only been found to occur in patients with decreased renal clearance and thus very high serum concentrations of the drug. Importantly, however, lactic acidosis may not always be coded in patients’ notes and hence its occurrence may be underestimated.

The most common side-effects of metformin are gastrointestinal, occurring most frequently at initiation of treatment, and usually resolve. If gastrointestinal side-effects do not resolve, a trial of a modified-release preparation should be initiated as this may improve tolerability and concordance.

Insulin secretagogues

Pancreatic insulin secretion is increased by both sulfonylureas and prandial glucose regulators (glinides). These agents bind to ATP-sensitive potassium channels in the pancreatic beta cells, which regulate insulin secretion. Channel closure and cell membrane depolarisation lead to an increase in cytoplasmic calcium resulting in increased insulin secretion, thus they are only effective when there is residual pancreatic function.

The most common side-effects are weight gain and hypoglycaemia. The risk of hypoglycaemia is highest in the elderly, in patients with renal dysfunction, if inadequate carbohydrate has been consumed, or after strenuous exercise or excess alcohol intake. Sulfonylureas can be used with caution in severe renal impairment and the lowest dose that achieves glycaemic control should be used. Patients should be counselled about hypoglycaemia and glucose monitoring equipment should be issued to at-risk patients.

Pioglitazone

The thiazolidinedione pioglitazone can be used as a first intensification of oral therapy at a dosage of 15–45mg once daily. Its mode of action is to bind to the nuclear receptor peroxisome proliferator-activated receptor (PPAR) gamma in adipose, muscle and liver tissue. This increases lipogenesis and enhances uptake of free fatty acids and glucose, which increases insulin sensitivity. Pioglitazone is eliminated mainly through the bile and can therefore be used in the presence of CKD.

Pioglitazone promotes sodium reabsorption in renal collecting ducts, which leads to fluid retention. Therefore, it should not be used in patients with heart failure or a past history of heart failure, and used with caution in patients with prior myocardial dysfunction.
Figure 1. NICE pathway for glucose management in type 2 diabetes. From: NICE Pathways. Type 2 diabetes in adults.

**Targets:**
Consider relaxing the target HbA1c on a case-by-case basis in:
- People who are older or frail
- People with significant co-morbidities such as cardiovascular disease or renal impairment

**Diet and lifestyle alone**

HbA1c >48mmol/mol

Metformin (modified-release if standard-release not tolerated)

HbA1c ≥58mmol/mol

**First intensification:**
- Metformin + DPP-4 inhibitor or
- Metformin + pioglitazone or
- Metformin + sulfonylurea or
- Metformin + SGLT2 inhibitor
Aim for HbA1c ≤53mmol/mol

HbA1c ≥58mmol/mol

**Second intensification:**
- Metformin + DPP-4 inhibitor + sulfonylurea or
- Metformin + pioglitazone + sulfonylurea or
- Metformin + pioglitazone + SGLT2 inhibitor or
- Metformin + sulfonylurea + SGLT2 inhibitor or
- Insulin-based treatment
Aim for HbA1c ≤53mmol/mol

HbA1c ≥58mmol/mol

**If metformin intolerant:**
- First line – DPP-4 inhibitor, pioglitazone or sulfonylurea
- Repaglinide can be considered, but outside current licence
- First intensification with:
  - DPP-4 inhibitor + pioglitazone or
  - DPP-4 inhibitor + sulfonylurea or
  - pioglitazone + sulfonylurea
- Second intensification with insulin-based treatment

If BMI ≥35 (33 in Asians), or BMI <35 for those in whom insulin would have occupational implications, or weight loss would benefit obesity related co-morbidities, choose metformin + sulfonylurea + GLP-1 agonist

If BMI <35 (33 in Asians), choose metformin + NPH insulin

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Figure 1. NICE pathway for glucose management in type 2 diabetes. From: NICE Pathways. Type 2 diabetes in adults.
Patients taking pioglitazone should be closely monitored for signs of heart failure.

Some studies suggest there may be a small increased risk of bladder cancer associated with pioglitazone use and so it should be avoided where there is a past history of bladder cancer or haematuria. There is also an increased fracture risk in the elderly, particularly postmenopausal women and patients with CKD, so prescribing should be limited for these groups.27

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**DPP-4 inhibitors**

Glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP) are incretin hormones that are stimulated by oral food intake, released into circulation and act to stimulate insulin secretion, slow gastric emptying and increase satiety. These effects lower postprandial blood glucose concentrations. GLP-1 and GIP are lysed by the enzyme dipeptidylpeptidase-4 (DPP-4). DPP-4 inhibitors therefore slow the breakdown of the incretins, permitting

<table>
<thead>
<tr>
<th>Drug</th>
<th>eGFR (ml/min/1.73m²)</th>
<th>15–29</th>
<th>30–59</th>
<th>60–89</th>
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<tbody>
<tr>
<td>Metformin</td>
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<tr>
<td>Sulfonylureas</td>
<td>Risk of hypoglycaemia with renal impairment</td>
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<tr>
<td>Pioglitazone</td>
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<tr>
<td><strong>DPP-4 inhibitors</strong></td>
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<tr>
<td>Alogliptin</td>
<td>Further dose reduction if eGFR &lt;30</td>
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<td>Linagliptin</td>
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<tr>
<td>Sitagliptin</td>
<td>Further dose reduction if eGFR &lt;30</td>
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<tr>
<td>Saxagliptin</td>
<td>Use with caution</td>
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<td>Vildagliptin</td>
<td>Use with caution</td>
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<td><strong>GLP-1 agonists</strong></td>
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<td>Dulaglutide</td>
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<td>Exenatide standard-release</td>
<td>Dose reduction if eGFR &lt;50</td>
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<td>Exenatide modified-release</td>
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<td>Liraglutide</td>
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<td>Lixisenatide</td>
<td>Use with caution</td>
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<tr>
<td><strong>SGLT2 inhibitors</strong></td>
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<tr>
<td>Canagliflozin</td>
<td>Avoid if eGFR &lt;45</td>
<td>Dose reduction if eGFR &lt;60</td>
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<tr>
<td>Dapagliflozin</td>
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<td>Empagliflozin</td>
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<tr>
<td><strong>Use freely</strong></td>
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<td><strong>Restricted use</strong></td>
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<tr>
<td><strong>Not recommended</strong></td>
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Table 2. Guidance on prescribing oral antihyperglycaemic agents for patients with renal impairment

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27. Patients taking pioglitazone should be closely monitored for signs of heart failure.
endogenous GLP-1 and GIP to exert their glucose-lowering effect for longer.  

The most common side-effects of DPP-4 inhibitors are gastrointestinal. There has been a reported association with acute pancreatitis, although this has not been confirmed.  

Most DPP-4 inhibitors are renally excreted and therefore require dose reductions for eGFR less than 50ml/min/1.73m². Linagliptin is the exception, as it is excreted in the bile, and can replace metformin as monotherapy if the eGFR declines below 30ml/min/1.73m².

GLP-1 agonists

GLP-1 agonists act via GLP-1 receptors to increase insulin secretion, suppress glucagon secretion, slow gastric emptying and increase satiety. They are given by subcutaneous injection and can be used in conjunction with oral antihyperglycaemic agents to achieve glycaemic control.

These agents can be used when the eGFR is greater than 30ml/min/1.73m² but a dose reduction is advised when the eGFR falls below 30ml/min/1.73m² for exenatide and lixisenatide. At the time of writing, we understand there is an application for licensing liraglutide down to an eGFR of 15ml/min/1.73m².

GLP-1 agonists are given by subcutaneous injection. Side-effects are most commonly gastrointestinal, usually occurring shortly after commencement of therapy, and subside over time. An association with acute pancreatitis is reported, although again not confirmed.

SGLT2 Inhibitors

Sodium-glucose co-transporter 2 (SGLT2) inhibitors, or gliflozins, appear to have some important renoprotective effects, possibly by improving glycaemic control, lowering blood pressure and via a direct effect on the nephrons. They act by reducing glucose and sodium reabsorption in the proximal tubule, causing tubuloglomerular feedback, afferent arteriolar vasoconstriction and reduction in hyperfiltration. In patients with type 2 diabetes, they give rise to a transient reduction in eGFR. Animal studies have demonstrated that SGLT2 inhibitors decrease glomerular inflammation and fibrosis, and reduce albuminuria. The results of an extension of the EMPA-REG OUTCOME trial demonstrated that empagliflozin had favourable renal outcomes in patients with severe renal impairment and diabetes, and this could encourage broader use of such drugs in patients with diabetic nephropathy in the future.

The most common side-effects of SGLT2 inhibitors are polyuria and increased risk of bacterial and fungal genitourinary infections. Canagliflozin has been associated with an increased risk of osteoporotic fractures and reduced bone mineral density and also an increased risk of minor amputation in the feet.

SGLT2 inhibitors are not licensed for commencement if the patient’s eGFR is below 60ml/min/1.73m². Dapagliflozin is not recommended in patients with an eGFR less than 60ml/min/1.73m², while empagliflozin and canagliflozin can be used down to an eGFR of 45ml/min/1.73m².

Insulin

Prescribing insulin for patients with CKD is safe if reviewed regularly and patients receive appropriate counselling about hypoglycaemia. A consensus approach does not exist on the choice of insulin in patients with diabetes and end-stage renal disease. Due to reduced renal excretion, patients with CKD are at higher risk of hypoglycaemia and dose adjustments will often be required in those with an eGFR less than 50ml/min/1.73m².

Dialysis

Patients who receive any type of renal replacement therapy should receive ongoing specialist diabetes management. Renal replacement therapy can affect glycaemic control via several mechanisms: by clearing glucoregulatory hormones (such as insulin and glucagon) and antidiabetic drugs; by changing insulin metabolism through causing fluctuations in uraemia, acidosis and phosphate metabolism; and by directly changing the blood glucose concentration via the dialysate. Glycaemic control on dialysis days can therefore be complex and unpredictable; data from continuous glucose monitoring can be used to help vary the insulin dosage around dialysis days.

Peritoneal dialysis can affect glycaemic control by similar mechanisms to those of haemodialysis, with the effect of glucose content of dialysate being more pronounced. In addition, some glucose monitors are affected by the non-glucose sugars in peritoneal dialysate and can give falsely elevated readings.

Transplantation

Post-transplant diabetes mellitus (PTDM) can affect between 10% and 75% of kidney transplant recipients, and confers a worse prognosis than those not affected. Advancing age, non-Caucasian ethnicity, presence of hepatitis C or cytomegalovirus, obesity, hyperlipidaemia, high-dose steroids and the use of calcineurin inhibitors increase the risk of developing PTDM. The condition should be treated in the same way as type 2 diabetes.

Conclusion

CKD is common in patients who have diabetes. Microalbuminuria is the first clinical indicator of diabetic nephropathy and should be monitored, alongside biochemical tests of renal function, so that prescribing of both oral antihyperglycaemic agents and other medication can be adjusted. Hypertension, smoking, hypercholesterolaemia, diet, exercise and glycaemic control are all modifiable risk factors and should be managed aggressively, taking into consideration the limitations of renal dysfunction on polypharmacy.

The armamentarium for prescribing in severe renal disease is limited to insulin, linagliptin and pioglitazone with cautious use of sulfonylureas. This may be extended to the use of SGLT2 inhibitors and liraglutide in the near future. Patients who have severe renal impairment, those who receive renal replacement therapy and those who develop PTDM present a wide variety of challenges relating to glycaemic control and should be managed by specialists.
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Declaration of interests
None to declare.

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