# Hyperkalaemia in CKD and diabetes

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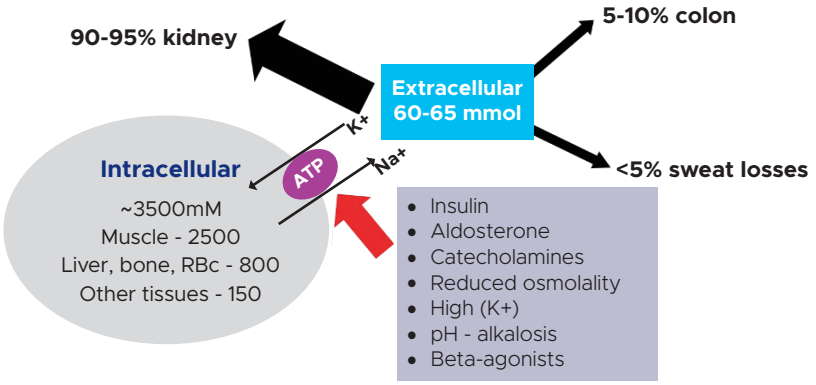
**Abstract**

Hyperkalaemia, both acute and chronic, limits the ability to optimally manage people with chronic kidney disease, cardiovascular disease and diabetes with renin angiotensin aldosterone system inhibitors. Data demonstrate the benefits of optimal doses of these drugs in delaying kidney, cardiac and diabetic kidney disease progression and reducing mortality. This review briefly summarises the strategies for hyperkalaemia management and the recommendations from the UK guidelines.

**Key words**: chronic kidney disease, diabetes, heart failure, hyperkalaemia, potassium binders, aldosterone

**Figure 1.** Potassium movement in the body




## Background

### Potassium homeostasis

Potassium homeostasis is essential for normal cellular functions through alteration of membrane excitability: muscle contraction, neuromuscular excitability and cardiac pacemaker rhythmicity. In addition, it helps to maintain cell volume and to control acid-base balance and DNA and protein synthesis.

Symptoms related to hyperkalaemia are usually few but may include neuromuscular disturbances such as muscle aches or lack of strength and weakness leading to a flaccid paralysis; tingling sensation; feeling nauseous and vomiting; palpitations and, most seriously, life-threatening ventricular arrhythmias and sudden cardiac death.[1]

Physiologically, the kidneys are responsible for more than 90% of the potassium filtered that is ingested each day (Figure 1).[2-4] Initially, potassium-rich foods which contain glucose cause a release of insulin, which drives potassium into cells.[4] The increase in dietary potassium also increases urinary and gastrointestinal elimination of potassium via a gut-kidney kaliuretic pathway.[5] The colon excretes potassium, and this can increase more than threefold in people with chronic kidney disease (CKD).[5] The majority of ingested potassium is filtered within the glomerulus, then reabsorbed by the proximal tubule and the loop of Henle, leaving approximately 10% to continue to the distal tubule. The secretion of excess potassium into the urine occurs within the principal cells of the renal collecting duct. This process is dependent on the luminal potassium channels which are impacted by the basolateral sodium/potassium adenosine triphosphatase pump ($Na^+/K^+$- ATPase), working in conjunction with a luminal sodium channel.[2, 3]

Potassium secretion in the collecting tubule is determined by serum aldosterone levels and sodium concentrations within the distal tubule.[1] Aldosterone stimulates potassium secretion by increasing the activity of the epithelial sodium channel (ENaC), which is electrochemically coupled to the renal outer medullary potassium-secreting (ROMK) channels. It also stimulates the movement of potassium into cells. The kidney itself is also capable of sensing an increase in plasma potassium and this leads to inhibition of the thiazide-sensitive sodium–chloride cotransporter (NCC) in the distal convoluted tubule.[2,3]

## The benefits of renin angiotensin aldosterone system inhibitors (RAASi) in clinical practice

RAASi therapy is recommended as the cornerstone of evidence-based guideline-directed care featured in cardiology, diabetes and nephrology guidelines (KDIGO, NICE, ESC and AHA amongst others). [6-8]

High-quality randomised clinical trials have emphatically demonstrated that RAASi reduce the risk of death and slow disease progression in people with heart failure (HF), CKD, and diabetes (DM). These studies,[9,10] in addition to cohort studies, have shown the value of optimising RAASi. A recent large database examining a UK-wide network of more than 1,100 primary care practices and linked to Hospital Episode Statistics (HES) data showed that use of maximal doses versus submaximal doses of RAASi in 100,572 patients with CKD (of any cause), and 12,113 patients with HF is associated with an increased mortality rate.[11] This real-world evidence has shown that the incidence of major adverse cardiac events (MACE) and mortality was consistently higher in the lower-dose group (adjusted incident rate ratios: CKD 5.60 [95% CI 5.29-5.93] for mortality and 1.60 [95% CI 1.55-1.66] for non-fatal major adverse cardiac events.[11] For heart failure the corresponding figures were 7.34 [95% CI 6.35-8.48] for mortality and 1.85 [95% CI 1.71-1.99] for major adverse cardiac events.

Even in patients with advanced CKD, the STOP ACEI trial confirmed that there was no renal benefit in discontinuing RAASi and indeed there was a trend to a reduction in requiring kidney

replacement therapy (KRT) in those maintained on RAASi therapy.[12] In addition, those who discontinued treatment had higher levels of proteinuria, a surrogate marker of poor outcomes. The guidelines specifically recommend that RAASi be titrated up to moderate to high doses, as used in clinical trials, to derive optimal treatment benefits.

Looking into the future of preserving kidney function, the SGLT2-i and GLP-1 receptor agonists are promising in delaying kidney progression and perhaps assisting in lowering potassium while the newer non-steroidal mineralocorticoid receptor antagonists (MRAs), endothelin antagonists and aldosterone synthase inhibitors may present further challenges in their use due to effects on serum potassium levels. Modelling suggests that use of all these treatments may lead to a 5-year saving of the need for dialysis.[13]

However, the greatest challenge in optimising drug therapy remains minimising the development of hyperkalaemia, which increases with worsening CKD.[14] When hyperkalaemia develops in these patient groups it is important to recognise the potential causes and to treat appropriately.

Considering acute hyperkalaemia and its management, six main scenarios are possible:

1. Spurious or pseudohyperkalaemia
2. Increased intake of potassium
3. Cellular movement of potassium
4. Reduced loss or removal of potassium
5. Treatment-related hyperkalaemia caused by medications (e.g. RAASi, MRAs, non-steroidal anti-inflammatory drugs [NSAIDs], diuretic agents, heparin)
6. Possible adrenal insufficiency and hyporeninaemic hypoaldosteronism if clinically relevant

Spurious hyperkalaemia is often due to a traumatic venepuncture, a tight tourniquet or delay in sample processing, which can raise the plasma potassium by 0.5 to 1.0 mmol/L. Haematological disorders such as haemolysis, thrombocytosis and leucocytosis or hereditary spherocytosis causing a large cell turnover can also lead to it. If pseudohyperkalaemia is suspected, the recommendation is to send paired blood samples in a clotted tube (serum) and a lithium heparin tube (plasma). Pseudohyperkalaemia is present if [serum $K^+$] – [plasma $K^+$] > 0.4mmol/L.[15]

Excess dietary intake of foods high in potassium or sodium supplements containing high potassium content can cause hyperkalaemia. More commonly hyperkalaemia can be precipitated by using solutions such as Hartmann's or Ringers lactate which contain 4-5 mmol/L of potassium. Finally, numerous drugs impact potassium concentrations via effects on renin, aldosterone and renal loss, as summarised in Table 1.

Potassium is redistributed from tissue catabolism from surgery, tissue necrosis as in rhabdomyolysis, burns, tumour lysis and by the effects of several medications (Figure 1). Hyperkalaemia can also occur in the setting of metabolic acidosis, in which a reduction in pH decreases sodium–hydrogen exchange which leads to a decrease in intracellular sodium and $Na^+/K^+$- ATPase activity. Rare causes of hyperkalaemia due to redistribution include hyperkalaemic periodic paralysis (an inherited channelopathy) or digoxin overdose (due to inhibition of $Na^+/K^+$-ATPase).

**Table 1.** Drugs that may cause hyperkalaemia

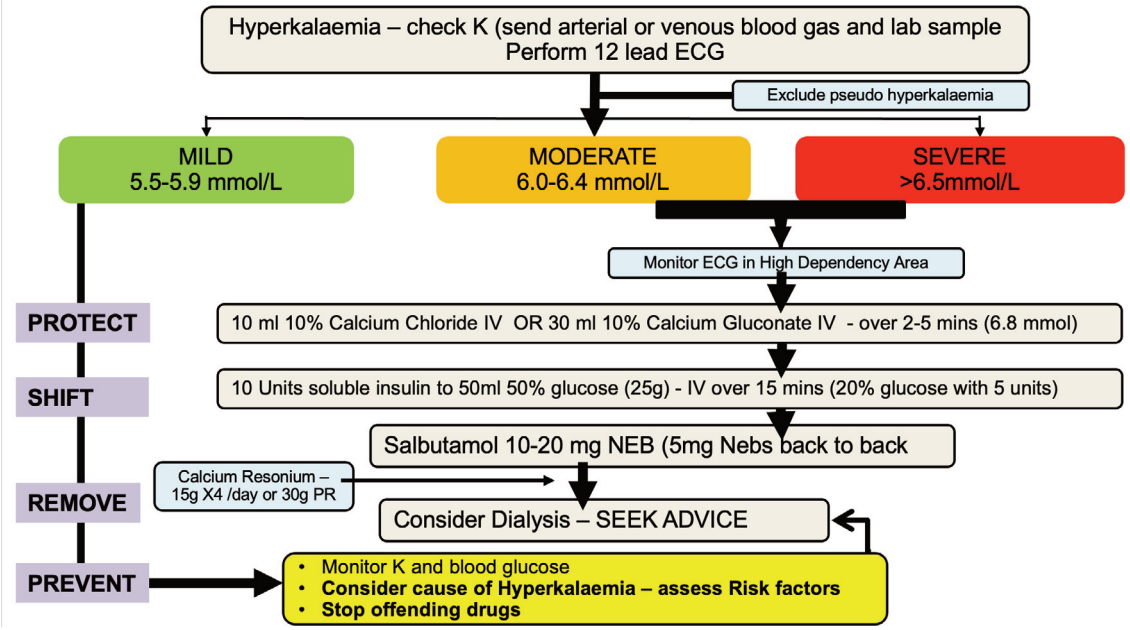
| Impaired release of renin or inhibitor or renin | Angiotensin converting enzyme inhibitor or angiotensin receptor blocker | Impaired aldosterone mechanism or blockage | Actions on tubules and channels e.g. sodium channel blockage and potassium movement from cells |
|---|---|---|---|
| NSAIDs | Ramipril | Adrenal insufficiency | Amiloride, triamterene |
| Beta blockers | Enalapril, perindopril, benazepril, quinapril, fosinopril, trandolapril, captopril | Heparin – inhibits aldosterone synthase | Pentamidine, trimethoprim |
| Calcineurin inhibitors: ciclosporin, tacrolimus | Irbesartan | Ketoconazole | Succinylcholine – K release from cells |
| Renin inhibitor aliskiren | Candesartan, olmesartan, telmisartan, eprosartan, valsartan, losartan | Spironolactone, epleronone, finerenone | Aldosterone synthase inhibitors - decreasing both genomic and non-genomic effects of aldosterone: osilodrostat, baxdrostat, lorundrostat |
| | | Angiotensin receptor–neprilysin inhibitors (ARNi) sacubitril-valsartan | Digoxin |
| | | | Arginine – moves K out of cells |

Reduced urinary potassium excretion may occur with all causes of acute kidney injury leading to hyperkalaemia, especially if this is accompanied by oliguria or anuria. In CKD, even a severely reduced glomerular filtration rate still allows for the filtration of a considerable amount of potassium but the rate-limiting step in hyperkalaemia is usually potassium secretion by the tubules.

In addition to kidney disease, RAASi can block aldosterone effects, leading to hyperkalaemia. Hyperkalaemia may also be seen due to high serum aldosterone in HF patients. This may cause increased sodium reabsorption in the proximal tubule, leading to decreased sodium delivery to the distal nephron and downregulating potassium secretion. Co-morbid renal disease may disrupt renin production, shutting down the RAAS and leading to decreased aldosterone and potassium elimination.

When acute hyperkalaemia is established, treatment is based on the level of potassium, defined as: mild (5.0–5.5 mmol/L), moderate (5.6–6.0 mmol/L) and severe (>6.0 mmol/L).[16] The threshold at which hyperkalaemia poses a risk of life-threatening arrhythmias or death is not consistent. Therefore, it is advisable to perform an ECG initially. Emergency management is indicated in

**Figure 2.** Acute management algorithm for hyperkalaemia




patients with ECG changes and/or potassium levels >6.5 mmol/L. First, intravenous calcium gluconate or calcium chloride is given to stabilise the myocardium and prevent life-threatening arrhythmias (Figure 2). Serum potassium is lowered quickly by administering glucose with insulin and/or β-2 adreno-receptor agonists, which redistribute potassium from the extracellular to the intracellular space. However, this is a temporary measure lasting up to four hours.

To eliminate potassium from the body, three routes can be adopted: excretion from the urine with the help of potassium-wasting diuretics like loop or thiazide diuretic agents; removal from the blood via haemodialysis, although this is only done in severe kidney insufficiency or when the patient is refractory to other methods; and, lastly, elimination via the gastrointestinal tract by using potassium binders.[16-18] In patients with hyper-kalaemia and concomitant metabolic acidosis, often seen in CKD, sodium bicarbonate is effective in lowering potassium levels.[16]

Trials have shown the value of potassium binders in the management of both acute and chronic hyperkalaemia and allowing the optimal use medications such as RAASi drugs (see later). On average, in open-label studies, during the induction phase of 2-4 hours, the use of sodium zirconium cyclosilicate (SZC) results in up to a 0.5 mmol/L fall in serum potassium concentrations within a few hours.[17]

## Chronic hyperkalaemia

The management of chronic hyperkalaemia is critical to allow the optimal use of RAASi therapy. The overriding emphasis is a holistic approach, reducing these beneficial drugs only as a last resort when other measures fail. It can be broken down into several interventions.

1. Dietary measures
2. Drugs that may compromise kidney function
3. Sodium bicarbonate
4. Diuretics
5. Potassium binders

First, dietary measures to limit the intake of foods rich in bioavailable potassium (e.g. processed foods) to <2.4 g/day for people with CKD G3–G5 who have a history of hyperkalaemia or as a prevention strategy during disease periods, as suggested by the UKKA guideline (Table 2), after non-dietary causes of hyperkalaemia have been addressed.[19] This remains challenging since higher potassium concentrations are associated with a decreased incidence of stroke, improved bone health, reduced blood pressure and reduced risk of nephrolithiasis.[20] Indeed, KDIGO has focused on processed foods, which have a 90% absorption rate, compared to plant-based foods which have an absorption rate of 50-60% and animal-based foods 70-90%.

Second are drugs that may compromise renal function and increase potassium levels, such as NSAIDs. These should be stopped either permanently or temporarily.

Bicarbonate therapy is used to assist in the intracellular shift of potassium. However, the evidence is limited and heterogeneous. In two long-term studies (longer than two months), alkali therapy was shown to be associated either with a significant net decrease in serum $K^+$ by approximately 0.7 mmol/L or no significant change, as was shown in short-term studies (≤7 days).[16] This measure remains in clinical practice and guidelines suggest treatment with bicarbonate if the bicarbonate level is <22 mmol/L with or without hyper-kalaemia in the UKKA guideline; <18 mmol/L in the KDIGO guideline or

**Table 2.** UKKA guidelines on management of hyperkalaemia

| Guideline | Recommendation | Grade |
|---|---|---|
| 1.2.1 | We recommend that the serum $K^+$ is repeated within 3 days, or as soon as feasible, if an episode of mild hyperkalaemia ($K^+$ 5.5 – 5.9 mmol/L) is detected unexpectedly in the community | 1C |
| 1.2.2 | We recommend that the serum $K^+$ is repeated within 1 day of an episode of moderate hyperkalaemia (K+ 6.0 – 6.4 mmol/L) when detected in the community | 1C |
| 1.2.3 | We recommend that patients with severe hyperkalaemia ($K^+ \geq$6.5 mmol/L) detected in the community are admitted for immediate assessment and treatment | 1B |
| 2.1 | We recommend that urea and electrolytes should be assessed prior to initiation of ACE-I or ARB and these drugs should be used with caution if the serum $K^+$ is >5.0 mmol/L | 1A |
| 2.2 | We suggest that initiation of MRAs should be avoided in patients with a baseline serum $K^+$ >5.0 mmol/L or eGFR <30 ml/min | 1B |
| 5.1 | We recommend that dietary strategies to modify potassium intake are instituted for patients with CKD and persistent hyperkalaemia with a serum $K^+$ >5.5 mmol/L after non-dietary causes of hyperkalaemia (constipation, acidosis and poorly controlled diabetes) have been addressed | 1B |
| 7.1 | We suggest that loop diuretics may be a useful adjunct for the treatment of chronic hyperkalaemia in patients who are non-oliguric and volume replete | 2C |
| 2.5 | We suggest increased frequency of monitoring in patients with a serum $K^+$ between 5.5-5.9 mmol/L and consideration of dose reduction of RAASi drugs (ACE-I, ARB, MRA) | 1B |
| 2.6 | We recommend that RAASi drugs be withheld during acute intercurrent illness (e.g. sepsis, hypovolaemia and/or AKI) at all severities of hyperkalaemia | 1D |
| 2.7 | We recommend cessation of RAASi drugs in patients with serum $K^+ \geq$6 mmol/L who do not meet the criteria for treatment with patiromer or sodium zirconium cyclosilicate | 1B |

Key: ACE-I=angiotensin converting enzyme inhibitor; ARB-angiotensin receptor blocker; MRAs=mineralocorticoid receptor antagonists; CKD=chronic kidney disease; RAASi=renin angiotensin aldosterone system inhibitor; AKI=acute kidney injury

<20 mmol/L in the NICE guidelines.[16,19]

Diuretic therapy has a place in the management of chronic hyperkalaemia in patients who are normovolaemic or hypervolaemic and non-oliguric (Table 2). However, it is imperative to adhere to the 'sick day rules': diuretics should be withheld during acute illness.

UKKA (endorsed by NICE) recommends the use of potassium binders (sodium zirconium or patiromer) in the treatment of chronic hyperkalaemia.[16] Their current criteria consist of people with CKD Stage 3b-5 or heart failure and a serum $K^+ \geq$6.0 mmol/L and receiving a sub-optimal dose or not taking RAASi due to hyperkalaemia, and not on dialysis. These agents should be initiated by a specialist in secondary care but can be continued in primary care. They should be withdrawn if RAAS inhibitors are no longer suitable or discontinued.

The recent availability of gastrointestinal potassium binders may allow RAASi therapy optimisation, possibly decreasing the need for dose reduction or discontinuation.[21,22] Patiromer (patiromer sorbitex calcium) and SZC (sodium zirconium cyclosilicate) remove potassium by exchanging cations (calcium for patiromer, and sodium and hydrogen for SZC) for potassium in the gastrointestinal tract, thus increasing its faecal excretion. A number of clinical trials of patiromer administered once daily showed a decrease in baseline potassium levels at four weeks and up until 52 weeks, allowing patients to maintain RAASi therapy regardless of hyperkalaemia.[23,24] Sodium zirconium cyclosilicate in the HARMONIZE trial of 258 ambulatory patients with hyperkalaemia resulted in normokalaemia in 98% of patients at 48 hours and maintenance of normokalaemia with a once-daily dose of SZC, versus placebo.[17] A recent single-arm study assessing the efficacy of SZC in long-term treatment and maintenance therapy of hyperkalaemia in 751 CKD patients demonstrated that SZC corrects hyperkalaemia and maintains normokalaemia in outpatients, regardless of CKD stage.[25] The limiting factor with these binders is adverse events such as diarrhoea, which limit their use.

Finally, temporary interruption of RAASi therapy and following sick day rules may be warranted, but it is imperative to minimise the duration and to reinstate treatment swiftly while monitoring serum potassium levels.[16,26]

## Conclusion

Hyperkalaemia can be harmful in the acute setting because of its effects on the cardiac membrane potential and increasing arrhythmia risk. However, a greater concern might be the impact of hyperkalaemia leading to the decrease or discontinuation of RAASi therapy, which has a well-established mortality benefit. Novel potassium binders may play an important role in optimising potassium levels without compromising on life-preserving medications.



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