Utilization of Potassium Binders for the Management of Hyperkalemia in Chronic Kidney Disease: A Position Statement by US Nephrologists

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Two potassium (K^+) binders—patiromer sorbitex calcium and sodium zirconium cyclosilicate—are recommended by international guidelines for the management of hyperkalemia. There is, however, no universally accepted best practice for how to appropriately utilize K^+ binders in the long-term clinical management of CKD. A panel of eight US-based nephrologists convened in October 2022 to develop a consensus statement regarding utilizing K^+ binders in clinical practice to help manage patients with nonemergent, persistent/recurrent hyperkalemia in CKD. Consensus was reached on the following topics: (1) identifying risk factors for hyperkalemia; (2) serum K^+ monitoring before and during K^+ binder use; (3) utilizing K^+ binders in patients receiving renin-angiotensin-aldosterone system inhibitors and dialysis; and (4) when to initiate K^+ binders and their duration of use. These consensus statements for the use of K^+ binders may assist the nephrology community in optimizing management of hyperkalemia in patients across the spectrum of CKD.

Key Words: Chronic kidney disease, Consensus, Hyperkalemia, Potassium binder, Sodium zirconium cyclosilicate

Hyperkalemia (elevated serum potassium [K^+]) is a potentially life-threatening electrolyte disorder associated with increased risk of adverse cardiovascular and kidney outcomes, including fatal cardiac arrhythmias, cardiac arrest, and mortality.[1–3] The cation-exchange resins, sodium polystyrene sulfonate and calcium polystyrene sulfonate, were approved over 60 years ago for the management of hyperkalemia. Although widely used, these agents are limited by poor tolerability (including poor palatability and serious gastrointestinal adverse effects), lack of randomized clinical trial evidence supporting long-term safety and efficacy, and lack of specificity regarding cation binding.[4–7] Given these issues, newer agents with more favorable benefit-risk profiles were developed to address hyperkalemia management with the goal of improving outcomes in patients with CKD.

In the last decade, two additional K^+ binders have emerged—patiromer sorbitex calcium (patiromer) and sodium zirconium cyclosilicate (SZC). Patiromer and SZC were approved in 2015 and 2018, respectively, for the treatment of adults with hyperkalemia.[8,9] Patiromer is a nonabsorbed cation-exchange polymer that contains a calcium-sorbitol counterion.[8] SZC is a nonabsorbed zirconium silicate that preferentially captures K^+ in exchange for hydrogen and sodium.[9] Both patiromer and SZC increase fecal K^+ excretion through binding of K^+ in the

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gastrointestinal tract, thereby lowering serum K^+.[8,9] The product profiles of patiromer and SZC are summarized in Table 1. Patiromer and SZC have demonstrated robust efficacy and safety in patients with hyperkalemia in numerous clinical trials.[10–19] There are several apparent benefits over cation-exchange resins, including a more rapid onset of action (significant reductions in serum K^+ with SZC were observed as early as 1 hour),[13,20] evidence of benefit and markedly improved tolerability in short- and longer-term clinical trials across various patient populations,[10–19] and the potential to optimize renin-angiotensin-aldosterone system inhibitor (RAASi) therapy (including the renin-angiotensin system inhibitors [RASis], angiotensin-converting enzyme [ACE] inhibitors, and angiotensin receptor blockers [ARBs], as well as mineralocorticoid receptor antagonists [MRAs]) to sustain cardiovascular and kidney protective benefits.[16,21]

The use of patiromer and SZC to manage hyperkalemia in CKD is specifically recommended by international evidence-based guidelines, including the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines,[7,22,23] as well as consensus statements.[24–26] Despite these recommendations, use of K^+ binders in nephrology outpatient practice remains surprisingly low[27,28]; one factor contributing to this may be knowledge gaps surrounding the most appropriate use of K^+ binders.[29] For example, there is a lack of consensus regarding the following: when K^+ binders should be initiated and their duration of use[30,31]; the timing and frequency of serum K^+ monitoring[31]; how K^+ binders might be used to facilitate the use or optimization of RAASi therapy[31]; and when to use K^+ binders in the dialysis setting and, if so, how to modify the dialysis prescription.[32] In addition, patients generally have limited awareness of the potential benefits to be gained from K^+ binders, including liberalization of K^+ intake, particularly in patients with type 2 diabetes or those on selected drugs (eg, warfarin) and in others whose conditions obligate dietary modification.[33]

Accordingly, a panel of US-based nephrologists convened in October 2022 to discuss the challenges and develop a consensus view surrounding the utilization of K^+ binders in real-world clinical practice.

CLINICAL SUMMARY

- Hyperkalemia is a potentially life-threatening electrolyte disorder associated with an increased risk of adverse cardiovascular and kidney outcomes and mortality.
- There is no universally accepted best practice for how to appropriately utilize potassium (K^+) binders in the long-term clinical management of CKD.
- A panel of eight US-based nephrologists convened to develop a consensus of optimal utilization of K^+ binders in patients with hyperkalemia in CKD, based on clinical evidence, international guidelines, and broad clinical experience.
- This consensus framework will assist the nephrology community in the long-term management of hyperkalemia in patients across the spectrum of CKD.

METHODS

A panel of eight US-based nephrologists convened for a virtual meeting on October 19, 2022, with the aim of developing consensus guidance for physicians regarding the use of K^+ binders in patients with CKD and nonemergent (ie, not requiring emergency care), persistent/recurrent hyperkalemia. This report provides the panel's recommendations, based on a combination of published evidence and clinical experience. The questions discussed by the panel are shown in Table 2.

CONSENSUS GUIDANCE

Consensus guidance from the panel is summarized in Table 3. The proposed frameworks for monitoring serum K^+ and initiation/use of K^+ binders are described in Figs 1 and 2.

How Do We Prioritize Patients for Hyperkalemia Management With K^+ Binders Based on Their Risk Profile?

Risk factors for the development of hyperkalemia may be intrinsic (patient-specific) or extrinsic (eg, treatment- or diet-related).[7] We suggest that, when assessing patients with CKD, the identification of risk factors for hyperkalemia should be based on comprehensive clinical observation including intrinsic factors, such as advanced age, prior hyperkalemia (with consideration of the underlying cause, eg, a provoked or unprovoked event), and comorbidities (eg, declining kidney function, cardiovascular disease, uncontrolled diabetes, and metabolic acidosis), and extrinsic factors, such as concurrent medications (in particular, optimizing RASi therapy or adding an MRA and use of antihypertensive therapies), solid organ transplant, and diet.

How Often Should Serum K^+ Concentrations Be Monitored in Patients at High Risk of Hyperkalemia?

Despite recommendations, there is no consensus on the timing and frequency of serum K^+ monitoring in patients at risk of hyperkalemia.[31] Moreover, frequency of serum K^+ monitoring remains low in clinical practice and often does not meet guideline recommendations.[26,34,35]

We endorse the KDIGO recommendations for CKD with respect to the monitoring frequency of serum K^+ in at-risk patients[7,36]; the precise frequency of serum K^+ monitoring depends on CKD stage. Furthermore, any initiation/escalation in medications with the potential to increase hyperkalemia risk and/or clinical deterioration should prompt more frequent monitoring of serum K^+ concentration, ideally within 2 weeks of the event.[7,22,23,36] In patients receiving dialysis, serum K^+ monitoring at least monthly is considered appropriate, particularly following the long interdialytic interval. More frequent monitoring of serum K^+ is recommended in patients receiving dialysis who have widely variable serum K^+ concentrations.[32]

Table 1. Product Profiles of Patiromer Sorbitex Calcium and Sodium Zirconium Cyclosilicate

Product	Indication	Approval (US)	Description	Posology	Contraindications and Warnings	RCT Data
Patiromer sorbitex calcium[8]	Hyperkalemia Not for emergent, life-threatening hyperkalemia (due to delayed onset of action)*	2015	Nonabsorbed organic cation-exchange polymer containing a calcium-sorbitol counterion Increases fecal K^+ excretion through binding of K^+ for Ca^{2+} ions in the GI lumen, providing reduction of systemic serum K^+ concentrations Time to onset of action: 7 hours	Amorphous powder for suspension in water for oral administration Initial dose 8.4 g QD; increase dose as needed at ≥1-week intervals in 8.4-g increments	*Contraindications:* Hypersensitivity to patiromer or any of its components *Warnings:* GI AEs in cases of motility disorders Hypomagnesemia *Other observations:* Potential DDI; administer other oral medications at least 3 hours before or after patiromer administration	Pivotal RCTs conducted in patients with CKD, DKD, HF, and resistant hypertension
Sodium zirconium cyclosilicate[9]	Hyperkalemia Not for emergent, life-threatening hyperkalemia (due to delayed onset of action)*	2018	Nonabsorbed inorganic zirconium silicate that preferentially captures K^+ Increases fecal K^+ excretion through binding of K^+ and NH_4^+ for Na^{2+} and H^+ ions in the GI lumen, providing reduction of systemic serum K^+ concentrations Time to onset of action: 1 hour	Powder for suspension in water for oral administration Nondialysis: *Starting dose:* 10 g TID for up to 48 hours *Maintenance dose:* 10 g QD; if needed, uptitrate weekly in 5-g increments to achieve maintenance dose from 5 g QoD to 15 g QD Hemodialysis: *Starting dose:* 5 g QD on nondialysis days (consider 10 g QD if serum K^+ >6.5 mmol/L) *Maintenance dose:* From 5 to 15 g QD on nondialysis days	*Contraindications:* None *Warnings:* GI AEs in cases of motility disorders Edema (mild to moderate) Hypokalemia in hemodialysis *Other observations:* Potential DDI; administer other oral medications at least 2 hours before or after SZC administration. Small dose-dependent increase in serum bicarbonate concentration (significance unclear)	Pivotal RCTs conducted in patients with CKD, T2D, HF, and/or ESKD undergoing hemodialysis

Abbreviations: AE, adverse event; Ca^{2+}, calcium; DDI, drug-drug interaction; DKD, diabetic kidney disease; ESKD, end-stage kidney disease; GI, gastrointestinal; HF, heart failure; K^+, potassium; Na^{2+}, sodium; NH_4^+, ammonium; QD, once daily; QoD, every other day; RCT, randomized controlled trial; T2D, type 2 diabetes; TID, three times daily; SZC, sodium zirconium cyclosilicate.
*Not for use alone in emergent, life-threatening hyperkalemia, without standard of care.

Table 2. Topics Discussed by the Panel During the Virtual Advisory Board

Topic for Discussion
1. How do we prioritize patients for hyperkalemia management with K^+ binders based on their risk profile?
2. How often should K^+ concentrations be monitored in patients at high risk of hyperkalemia?
3. How should K^+ binders be utilized in patients receiving RAASi?
4. What is the optimum serum K^+ concentration for initiation of a K^+ binder?
5. How should K^+ binders be utilized in patients with end-stage kidney disease/receiving dialysis?
6. How should K^+ binders be used in the long term?
7. How often should K^+ concentrations be monitored in patients receiving K^+ binders?

Abbreviations: K^+, potassium; RAASi, renin-angiotensin-aldosterone system inhibitor.

Identification of hyperkalemia should prompt a review of any medications and a repeat test to measure serum K^+ concentration,[37] to determine whether management options have been successful and to provide feedback to the patient.

How Should K^+ Binders Be Utilized in Patients Receiving RAASi?

RASi (ACEi or ARB) are foundational therapies for the management of patients with CKD and heart failure.[22,23,36,38] International guidelines recommend RASi therapy at the maximum tolerated dose in patients with CKD and/or heart failure/diabetes to optimize treatment benefits in terms of reducing the risk of kidney failure and cardiovascular mortality and morbidity.[22,23] Furthermore, guidelines recommend that the nonsteroidal MRA finerenone can be added to maximum tolerated RASi therapy for treatment of CKD with type 2 diabetes.[22] Therapies that inhibit the renin-angiotensin-aldosterone system (ACEi, ARB, MRA, and direct renin inhibitors) have demonstrated pharmacodynamic K^+-sparing properties (eg, by limiting K^+ excretion) that increase the risk of hyperkalemia.[39–41] Careful attention should also be given to drugs that otherwise inhibit the epithelial sodium channel of the distal nephron (ie, amiloride, triamterene, and trimethoprim), which can contribute to hyperkalemia.

International clinical practice guidelines state that hyperkalemia (or risk of hyperkalemia) should not be a barrier to RAASi dose optimization and recommend measures to reduce serum K^+ concentration rather than dose reduction or cessation of RAASi therapy.[22,23] Management of patients with CKD and nonemergent, persistent/recurrent hyperkalemia therefore requires a systematic approach, initially toward treating correctable factors; eg, optimizing serum bicarbonate concentrations to correct metabolic acidosis, dietary modification, and the use of therapeutic options such as sodium-glucose cotransporter-2 (SGLT2) inhibitors or loop/thiazide diuretics.[42]

Dietary modification has its limitations, as there is only a weak relation between dietary K^+ intake and serum K^+ concentration in patients with CKD.[43] Moreover, dietary restriction relies on patient adherence and potentially deprives patients of "heart-healthy" foods.[7,31,42] Other conditions such as the use of K^+-sparing medications and metabolic acidosis are more likely to explain K^+ abnormalities.[42] Here, the initiation of a K^+ binder can be used to manage hyperkalemia, facilitating continued use of RAASi therapy and/or the provision of liberalized dietary restrictions.

SGLT2 inhibitors may be considered in patients with CKD at risk of hyperkalemia. These agents attenuate serum K^+ by inhibiting reabsorption of sodium at the proximal tubule,[44] with clinical evidence suggesting that SGLT2 inhibitors do not increase serum K^+.[45,46] Previous studies in patients with diabetes and high cardiovascular risk or with CKD have demonstrated that SGLT2 inhibitors may reduce the risk of hyperkalemia[47] and that the complementary mechanisms of action of SGLT2 inhibitors and RAASi provide incremental cardiovascular and kidney benefit.[48,49] It is unclear, however, if/how dose modification of RASi or MRA contributed to differences in hyperkalemia incidence.[50]

The use of loop diuretics or thiazide-like agents ("thiazides") in patients with CKD is generally limited by an unpredictable pharmacodynamic response, with effectiveness being dependent on the K^+-wasting potential of the drug and the degree of residual kidney function.[7,31] In addition, loop diuretics or thiazides can occasionally result in unfavorable metabolic complications, including hyponatremia, hyperuricemia, and hypocalcemia (with loop diuretics) or hypercalcemia (with thiazides).[51,52]

What Is the Optimum Serum K^+ Concentration for Initiation of a K^+ Binder?

After addressing correctable factors using the systematic approach for hyperkalemia management, we suggest that the magnitude of hyperkalemia that should trigger initiation of a K^+ binder in patients with CKD and persistent/recurrent hyperkalemia should be based on individual patient characteristics and clinical judgment (Table 3 and Fig 1). Physicians and other care providers should consider initiation of a K^+ binder in clinically stable patients with nondialysis-requiring CKD, whether they are receiving RAASi therapy or not, with a persistent/recurrent serum K^+ concentration of $\geq$5.0 mmol/L (Fig 2). Similarly, physicians and other care providers should **strongly** consider initiation of a K^+ binder in clinically stable patients with nondialysis-requiring CKD, whether they are receiving RAASi or not, with a persistent/recurrent serum K^+ concentration of $\geq$5.5 mmol/L, and also in those with a persistent/recurrent serum K^+ concentration of $\geq$5.0 mmol/L with a clinical requirement or event, eg, the need to initiate or downtitrate RASi to control hyperkalemia or in those with uncontrolled diabetes.

How Should K^+ Binders Be Utilized in Patients With End-Stage Kidney Disease/Receiving Dialysis?

There is currently no universally accepted consensus regarding best practice for the management of persistent/recurrent predialysis hyperkalemia, which occurs in

Table 3. Key Discussion Points and Consensus Guidance

Topic	Consensus Guidance
1. How do we prioritize patients for hyperkalemia management with K^+ binders based on their risk profile?	• Identification of risk factors for hyperkalemia should be based on comprehensive clinical observation, including intrinsic factors and extrinsic factors such as concurrent medications and diet. • *Patient-specific factors:* o Advanced age, K^+ intake (diet and supplements), prior hyperkalemia (with consideration of the underlying cause, eg, a provoked or unprovoked event). • *Comorbidities:* o CKD (Stage 3b or greater), dialysis, heart failure, poorly controlled type 2 diabetes, resistant hypertension (ie, requiring use of MRAs/diuretics), metabolic acidosis, solid organ transplant (requiring calcineurin inhibitors), type 4 renal tubular acidosis, any condition with an indication to optimize RASi, or to add a nonsteroidal MRA. • *Concurrent medications:* o RASi optimization; addition of a nonsteroidal MRA; use of an NSAID, nonselective beta blocker, calcineurin inhibitor, trimethoprim.
2. How often should K^+ concentrations be monitored in patients at high risk of hyperkalemia?	• Follow the KDIGO recommendations for CKD with respect to the monitoring frequency of serum K^+ in at-risk patients; frequency will depend on CKD stage. • Initiation/escalation in medications with the potential to increase hyperkalemia risk and/or clinical deterioration should prompt monitoring of serum K^+ concentration, ideally within 2 weeks. • Identification of hyperkalemia should initiate a review of any medications and a repeat test to measure serum K^+. • In patients receiving dialysis, serum K^+ monitoring at least monthly is considered appropriate, particularly following the LIDI, with more frequent monitoring recommended in patients with widely variable serum K^+.
3. How should K^+ binders be utilized in patients receiving RAASi?	• Management of patients with CKD and nonemergent, persistent/recurrent hyperkalemia should comprise a systematic approach based initially on management of correctable factors including diet, and understanding of the role of other potential medications. • K^+ binders can be used to manage hyperkalemia in patients with CKD, while facilitating continuation of RAASi therapy and/or the provision of liberalized dietary restrictions.
4. What is the optimum serum K^+ concentration for initiation of a K^+ binder?	• Correctable factors for hyperkalemia should first be addressed as per the systematic approach for hyperkalemia management. • Strength of the recommendation to initiate a K^+ binder (ie, consider or strongly consider) should be based on individual patient characteristics and the degree of hyperkalemia, as well as clinical judgment (Fig 2).
5. How should K^+ binders be utilized in patients with end-stage kidney disease/receiving dialysis?	• Initiation of a K^+ binder should be strongly considered among patients with predialysis serum K^+ concentration of $\geq$5.5 mmol/L or those who are candidates for a low dialysate K^+ ($<$2.0 mmol/L) (Fig 2). • Other circumstances may require utilization of K^+ binders. For example, if circumstances obligate a delay in initiation or reintroduction of dialysis (eg, in the setting of a newly placed peritoneal dialysis catheter or as a bridge if vascular access for hemodialysis cannot be established in a timely manner).

(*Continued*)

Table 3. Key Discussion Points and Consensus Guidance (*Continued*)

Topic	Consensus Guidance
6. How should K^+ binders be used in the long term?	• In some patients, K^+ binders should be used for as long as the underlying modifiable causes of hyperkalemia remain. • Duration of treatment will likely depend on clinical judgment and tolerability. • A one-size-fits-all approach to dosing is unrealistic; clinical judgment should guide individualized dosing of K^+ binders based on clinical factors including hyperkalemia severity and risk of adverse events.
7. How often should K^+ concentrations be monitored in patients receiving K^+ binders?	• The KDIGO recommendations are advocated for serum K^+ monitoring in patients with CKD, where monitoring frequency should be based on CKD stage and the frequency of clinic visits. • Clinical events to trigger increased serum K^+ monitoring include change of any medication that will likely affect serum K^+, evidence of acute kidney injury or infection, or risk of abrupt changes in serum K^+ concentration. • If possible, more frequent serum K^+ monitoring should be performed within 1-2 weeks, repeated weekly, and then repeated monthly if the desired target serum K^+ concentration is achieved.

Abbreviations: K^+, potassium; KDIGO, Kidney Disease: Improving Global Outcomes; LIDI, long interdialytic interval; MRA, mineralocorticoid receptor antagonist; NSAID, nonsteroidal anti-inflammatory drug; RAASi, renin-angiotensin-aldosterone system inhibitor; RASi, renin-angiotensin system inhibitor.

many patients who undergo maintenance dialysis.[32,53] In patients who undergo hemodialysis, eg, the use of a low dialysate K^+ (<2.0 mmol/L) can result in a steep K^+ gradient between elevated serum K^+ and low dialysate K^+, rapid extracellular K^+ shifts, and induction of cardiac arrhythmias, including sudden cardiac death.[7,54,55] There is ample evidence supporting the use of K^+ binders in the management of hyperkalemia in patients on dialysis. For example, in patients with hyperkalemia undergoing hemodialysis, SZC maintained a predialysis serum K^+ concentration of 4.0-5.0 mmol/L following the long interdialytic interval without the need for rescue therapy[56]; these findings led to the approval of SZC in patients receiving maintenance hemodialysis.[9]

In our experience, after addressing other correctable factors, physicians and other care providers should **strongly** consider initiation of a K^+ binder among patients receiving dialysis who have a persistent/recurrent serum K^+ concentration of ≥5.5 mmol/L during the long interdialytic interval or who require a low dialysate K^+ (<2.0 mmol/L) to correct hyperkalemia (Table 3 and Fig 2). Other circumstances may require utilization of K^+ binders (Table 3). For example, if circumstances obligate a delay in initiation or reintroduction of dialysis (eg, in the setting of a newly placed peritoneal dialysis catheter or as a bridge if vascular access for hemodialysis cannot be established in a timely manner). In patients with end-stage kidney disease who are not candidates for dialysis (eg, the very frail and/or those with multiple comorbidities), a K^+ binder might be initiated as conservative management to minimize the risk of hyperkalemia in the context of supportive care.[57]

How Should K^+ Binders Be Used in the Long Term?

Both patiromer and SZC have demonstrated long-term efficacy and tolerability for up to 1 year in clinical trials.[12,16] However, in the real-world setting, use of K^+ binders to manage hyperkalemia remains relatively low[27,28]; in one analysis in Japan, 5.8% of patients were receiving a K^+ binder at the time of the index hyperkalemic event and 24.0% received a K^+ binder during 1-year follow-up.[28] Furthermore, K^+ binders are often used transiently, ie, only until a hyperkalemia episode is resolved,[58] with recurrent hyperkalemia frequently observed following cessation of K^+ binders.[59] A report by the National Kidney Foundation surrounding best practices in the management of hyperkalemia advises that ongoing management is needed to correct the underlying disturbances in K^+ balance.[60] In some patients, K^+ binders should be used for as long as the underlying modifiable causes of hyperkalemia remain (Table 3 and Fig 1). Duration of treatment will likely depend on clinical judgment and tolerability.

The recommended starting dose as stated in the prescribing label for SZC is 10 g three times daily (up to 48 hours) and for patiromer is 8.4 g once daily, both adjusted based on serum K^+ concentration and desired target range.[8,9] While locally approved dosing and frequency should be adhered to, a one-size-fits-all target serum K^+ concentration is unrealistic in clinical practice; here, the panel recommends that an individualized dose will likely be ideal, based on clinical factors including severity of hyperkalemia and risk of adverse events.

How Often Should K^+ Concentrations Be Monitored in Patients Receiving K^+ Binders?

A number of consensus-based recommendations are available to guide serum K^+ monitoring following initiation of a K^+ binder.[26,29,30] Here, we specifically endorse the KDIGO recommendations,[7,22,36] where frequency of serum K^+

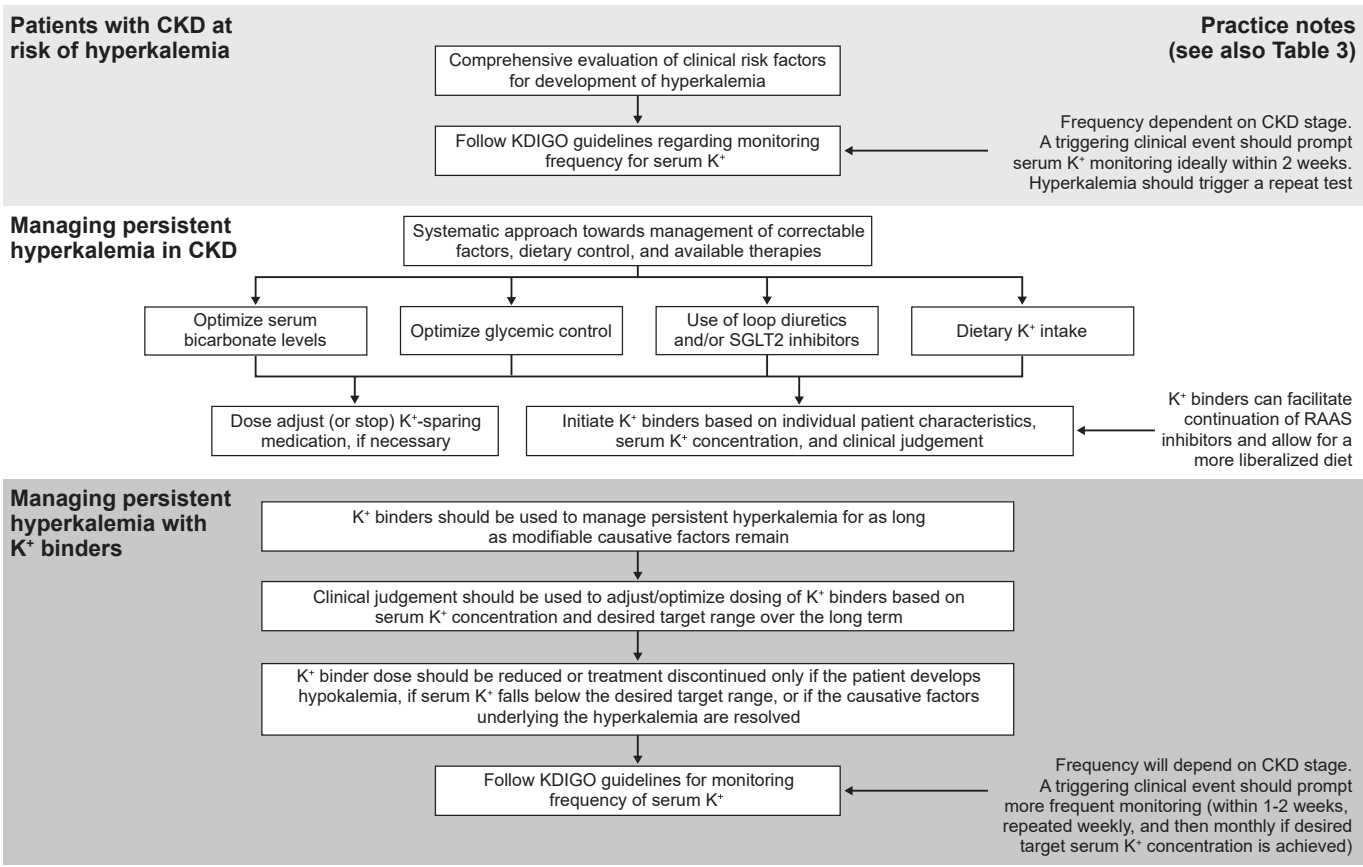

Figure 1. Framework for the use of K^+ binders in patients with CKD with nonemergent, persistent/recurrent hyperkalemia. Abbreviations: CKD, chronic kidney disease; K^+, potassium; KDIGO, Kidney Disease: Improving Global Outcomes; RAASi, renin-angiotensin-aldosterone system inhibitor; SGLT2, sodium-glucose cotransporter-2.

monitoring should be based on CKD stage and the frequency of clinic visits. Clinical events potentially triggering more frequent serum K^+ monitoring include changes in medication that can affect serum K^+ concentrations (eg, RASi initiation or uptitration, initiation of a nonsteroidal MRA, use of diuretics, change of K^+ binder or dose), or cessation of treatment with a K^+ binder, or change in electrolyte/volume status, as well as evidence of acute

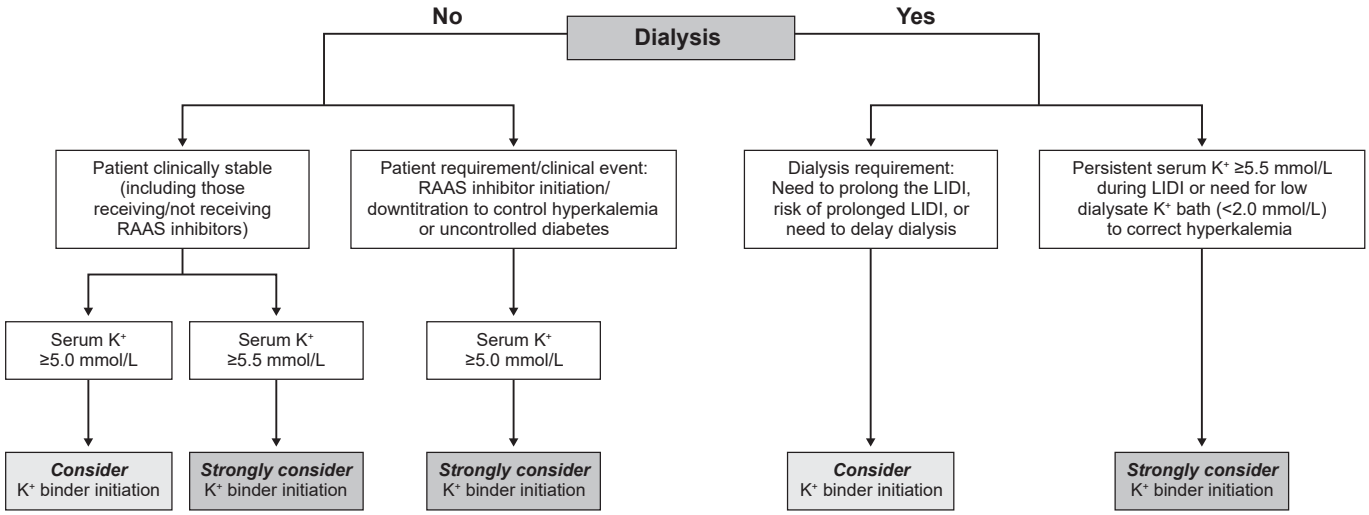

Figure 2. Proposed decision tree for initiation of a K^+ binder in patients with CKD with nonemergent, persistent/recurrent hyperkalemia. Abbreviations: CKD, chronic kidney disease; K^+, potassium; LIDI, long interdialytic interval; RAASi, renin-angiotensin-aldosterone system inhibitor.

kidney injury, intercurrent illness/infection, and risk of abrupt changes in serum K^+ concentration. A triggering clinical event should prompt more frequent monitoring (within 1-2 weeks, repeated weekly, and then repeated monthly if the target serum K^+ concentration is achieved).

CONCLUSIONS

This panel of nephrologists supports the use of K^+ binders in the long-term management of nonemergent, persistent/recurrent hyperkalemia, across the spectrum of CKD, including patients undergoing hemodialysis. The K^+ binders provide an effective and well-tolerated option for the management of hyperkalemia that can also facilitate continued and optimal use of RAASi therapy, which can help to slow CKD progression and improve the management of cardiovascular risk.

Our consensus positions are based on clinical evidence, international management guidelines, and broad clinical experience. Thus, we are providing a framework for the use of K^+ binders in the appropriate clinical setting and for monitoring serum K^+ postinitiation, which may assist the nephrology community in the optimization and long-term management of hyperkalemia in patients across the spectrum of CKD.

AUTHOR CONTRIBUTIONS

All authors attended the virtual advisory board and contributed to the drafting and revisions of the manuscript for important intellectual content. All authors approved the final version of the manuscript.

DATA SHARING STATEMENT

Data underlying the findings described in this manuscript may be requested in accordance with AstraZeneca's data sharing policy described at https://astrazenecagrouptrials.pharmacm.com/ST/Submission/Disclosure.

The AstraZeneca Group of Companies allows researchers to submit a request to access anonymized patient-level clinical data, aggregate clinical or genomics data (when available), and anonymized clinical study reports through the Vivli web-based data request platform.

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