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Restoring Balance From Hospital to Home: Updates on the Management of Acute and Chronic Hyperkalemia

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Supporter Acknowledgment

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Learning Objectives

- Discuss the importance of screening for, identifying, and assessing hyperkalemia
- Describe current prescribing information for novel therapies to the effective management of acute and chronic hyperkalemia
- Discuss recent safety and efficacy data supporting the use of novel therapies in patients with hyperkalemia
- Review multidisciplinary strategies for achieving optimal outcomes from hospitalization through discharge and outpatient follow-up

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Early Identification of Hyperkalemia

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Overview of Hyperkalemia

Etiology, Prevalence, and Mortality

Risk Factors and Potassium Homeostasis

The Importance of Early Diagnosis

HK Occurs in . . .

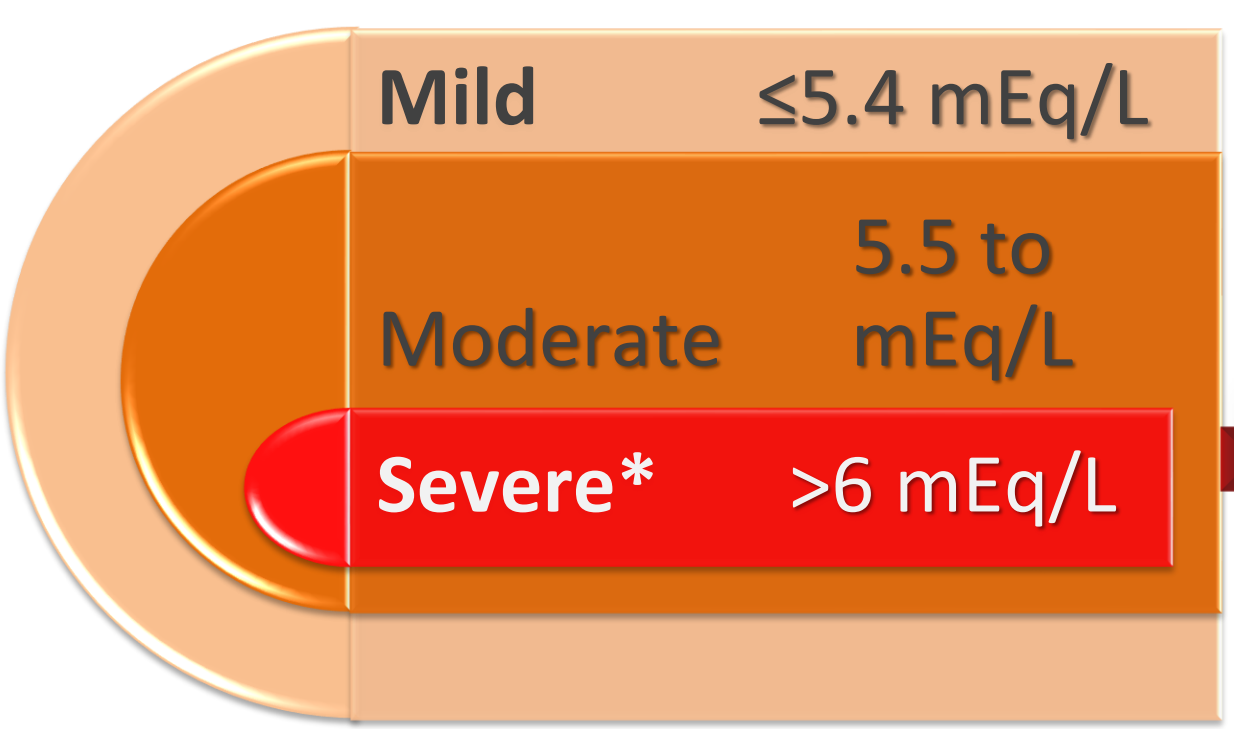
Up to 73% of patients with advanced CKD

. . . And up to 40% of patients with chronic HF

HK associated with increased hospitalizations and mortality—especially when K^+ testing and monitoring is not performed frequently

CKD, chronic kidney disease; HF, heart failure; HK, hyperkalemia.

HK Severity as Mortality Predictor



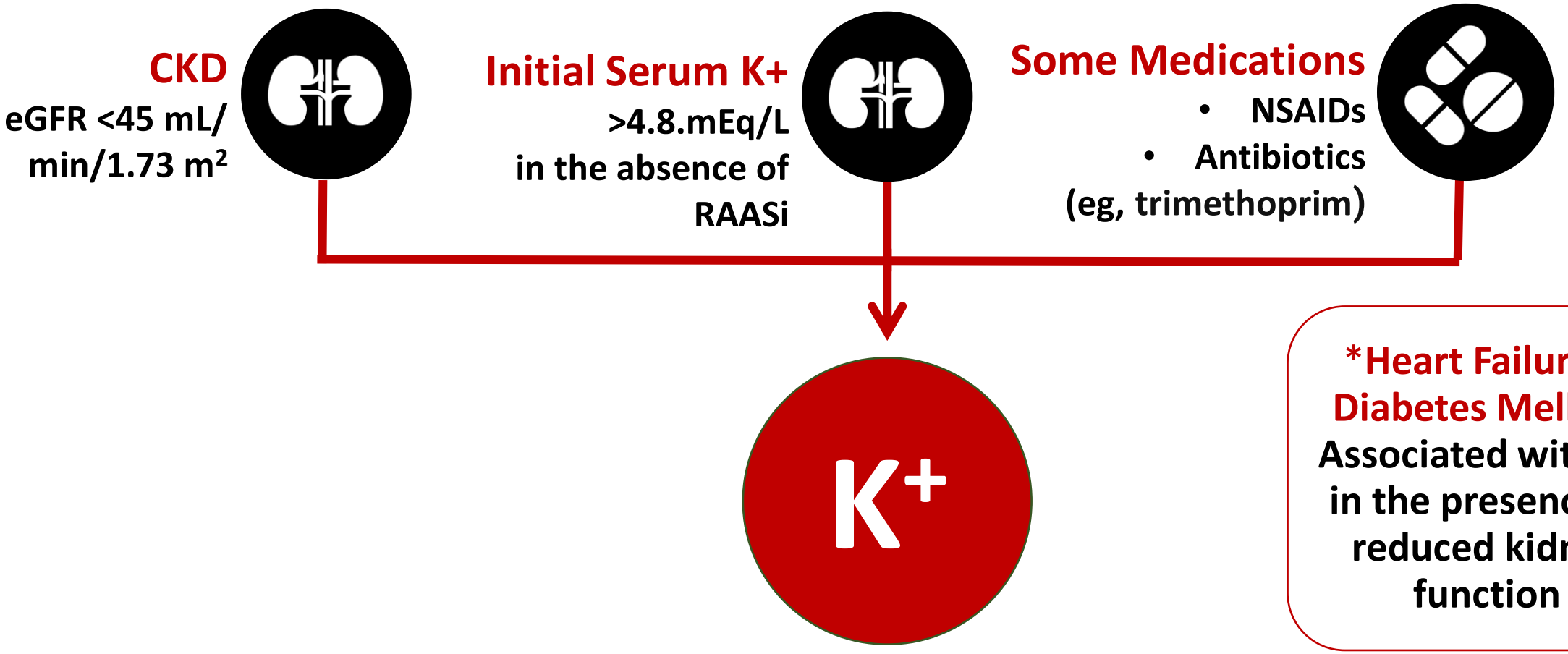
Severe HK is an independent predictor of all-cause and in-hospital mortality

Mortality rate is ~30%, mostly due to arrhythmias leading to cardiac arrest

*Not all sources agree with these cut-points; some put severe hyperkalemia at ≥7 mEqL.

Rosano GMC, et al: coordinated by the Working Group on Cardiovascular Pharmacotherapy of the European Society of Cardiology. *Eur Heart J*. 2018;4:180-188.

Risk Factors for HK*



***Heart Failure & Diabetes Mellitus** Associated with HK in the presence of reduced kidney function

eGFR, estimated glomerular filtration rate; NSAIDS, nonsteroidal anti-inflammatory drugs; RAASi, renin-angiotensin-aldosterone system inhibitors.

Lazich I, Bakris GL. *Semin Nephrol*. 2014;34(3):333-339. doi:10.1016/j.semnephrol.2014.04.008; Tromp J, et al. *Eur Heart J.* 2019;21(Suppl A):A6-A11.

Causes of HK

Pseudohyperkalemia

K^+ can be released by:

- Phlebotomy
- Fist clenching
- Incorrect tourniquet application

K^+ Cellular Shift/Redistribution

- Mineralacidosis
- Hypertonicity
- Insulin deficiency
- Beta blockers
- Alpha adrenergics
- Tissue injury
- Strenuous exercise

Excessive K^+ Intake

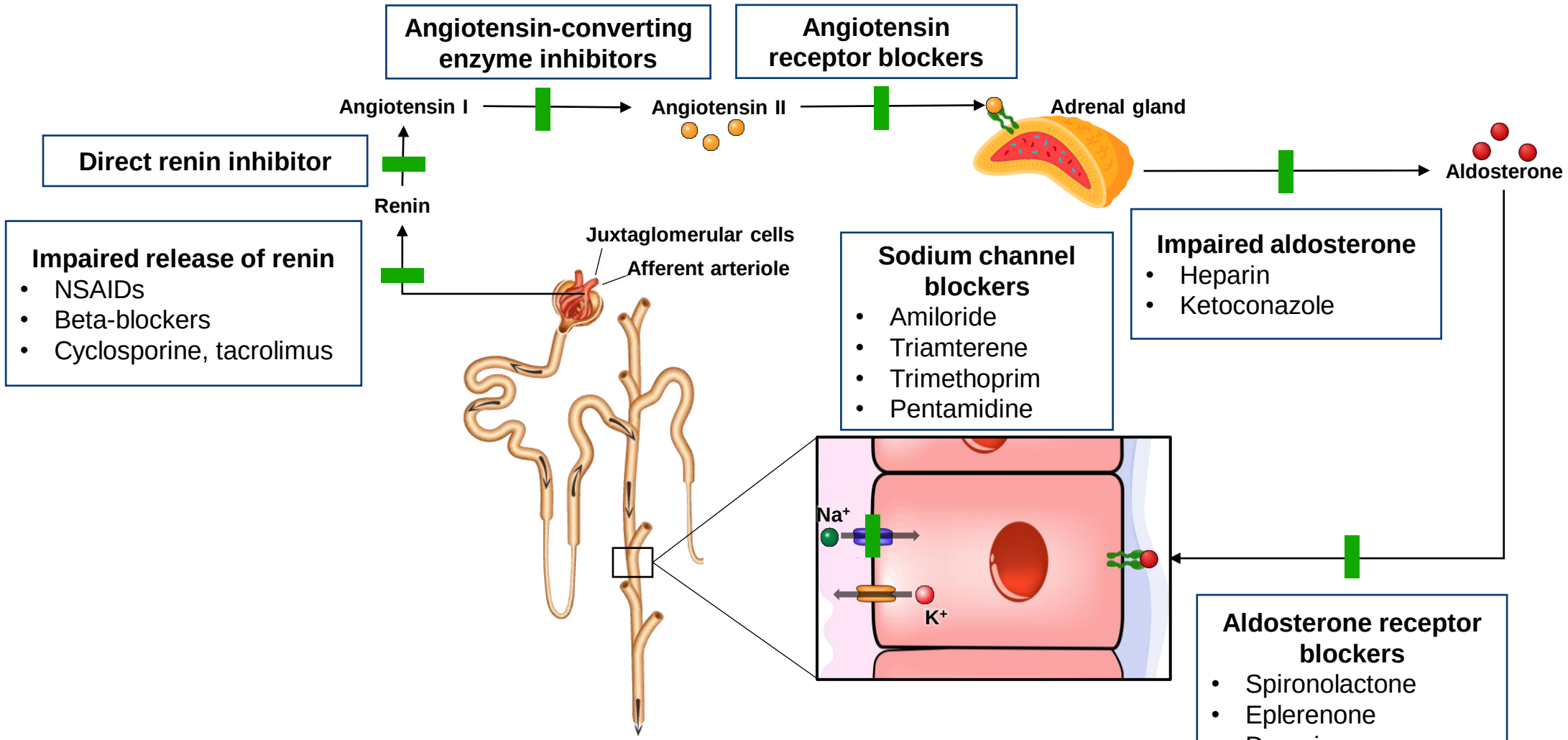
- Salt substitutes
- K^+ supplements
- High intake of food rich in K^+

Decreased Renal Excretion

- Decreased distal delivery of Na^+
- Mineral-corticoid deficiency

Palmer BF, Clegg DJ. *Adv Physiol Educ.* 2016;40:480-490; Palmer BF, Clegg DJ. *Cleve Clin J Med.* 2017;84:934-942.

Drugs that Interfere With RAAS



Adapted from: Clase CM, et al. *Kidney Int*. 2020:97:42-61. Bakris GL, et al. *N Engl J Med*. 2020;383(23):2219-2229. doi:10.1056/NEJMoa2025845

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Hyperkalemia Signs and Symptoms

Often Nonspecific, Common Symptoms: When to Suspect HK

Diagnosis and Monitoring: ECG Findings

HK Definition: Signs and Symptoms

HK: A common electrolyte imbalance marked by elevated serum potassium of (K^+) >5.5 mEq/L in patients with kidney disease

Acute in-hospital presentations can include any of these

Often, patients have *no symptoms* and the disorder can be chronic

Conduction abnormalities/cardiac arrhythmias/ (eg, sinus bradycardia, QRS prolongation, AV block, bundle block, ventricular tachycardia, fibrillation [less common])

Muscle weakness, possible paralysis

Fatigue, vomiting

De Nicola L, et al. *J Nephrol.* 2018;31:653-664; Littmann L, Gibbs MA. *J Electrocardiol.* 2018;51:814-817; Rosano GMC, et al. *Eur Heart J.* 2018;4:180-188.

Diagnosis and Monitoring

Identifying HK can be difficult:	• Patients are often asymptomatic • K^+ levels are dynamic
Suspect HK in patients with DM, HF, and/or CKD, especially if on RAASi	• ie, ARBs, ACEIs, and MRAs
Recommended in-hospital monitoring:	• Measure K^+ levels frequently after HK is identified and treatment started • Use continuous ECG for severe HK
Impact of long-term K+ monitoring on prognosis evaluated in large study of postdischarge HF patients	• Single baseline assessment insufficient, this neglects dynamic K+ changes over time • Long-term monitoring and K+ normalization can reduce HK-related AEs

ACEI, angiotensin-converting enzyme inhibitors; ARBs, angiotensin type II receptor blocker; DM, diabetes mellitus; ECG, electrocardiogram; MRA, mineralocorticoid receptor antagonists.

Palmer BF, Clegg DJ. *Cleve Clin J Med.* 2017;84:934-942; Núñez J, et al. *Circulation.* 2018;137:1320-1330.

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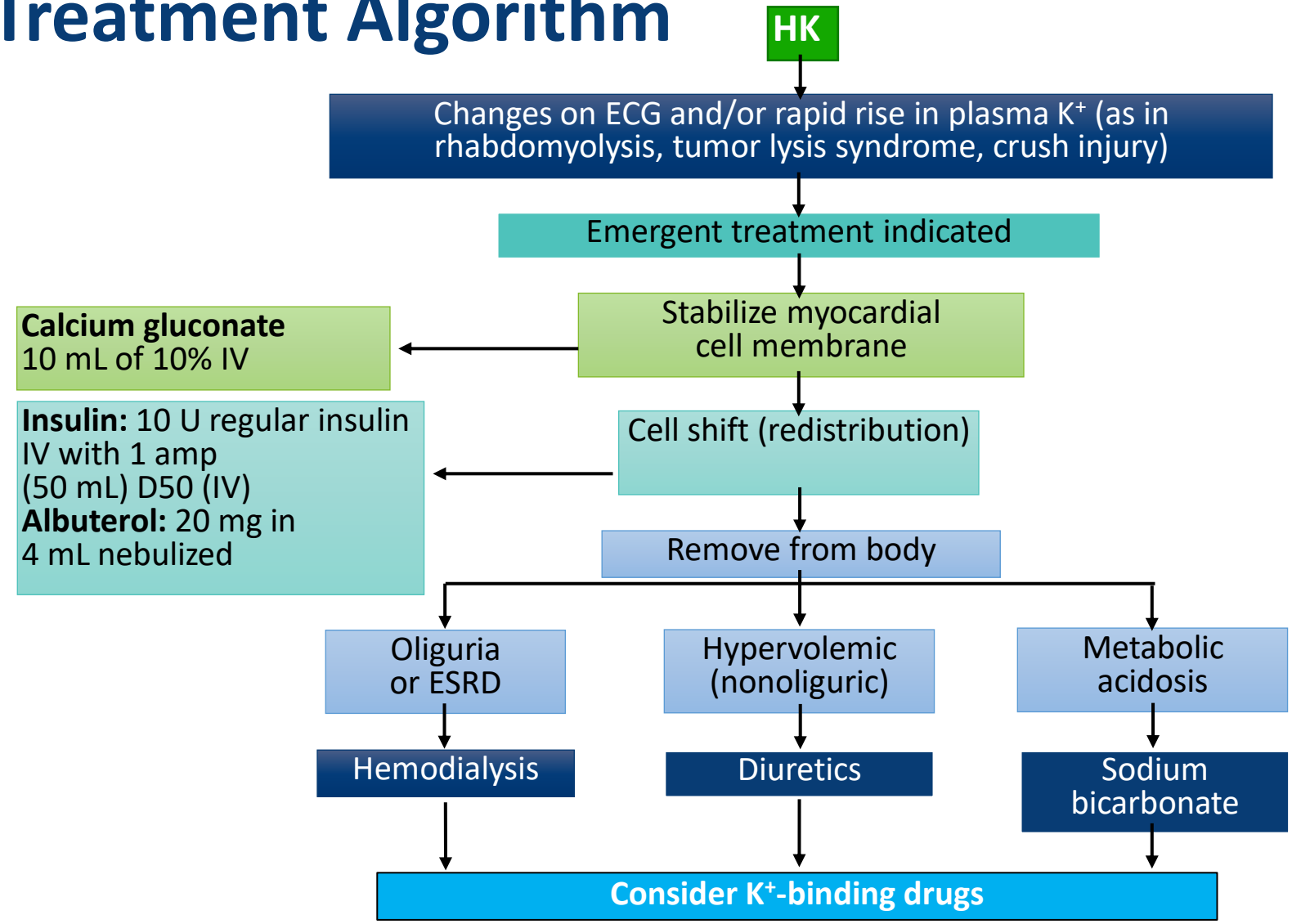
Identifying the Cause of Hyperkalemia

Etiological Algorithm for Evaluation

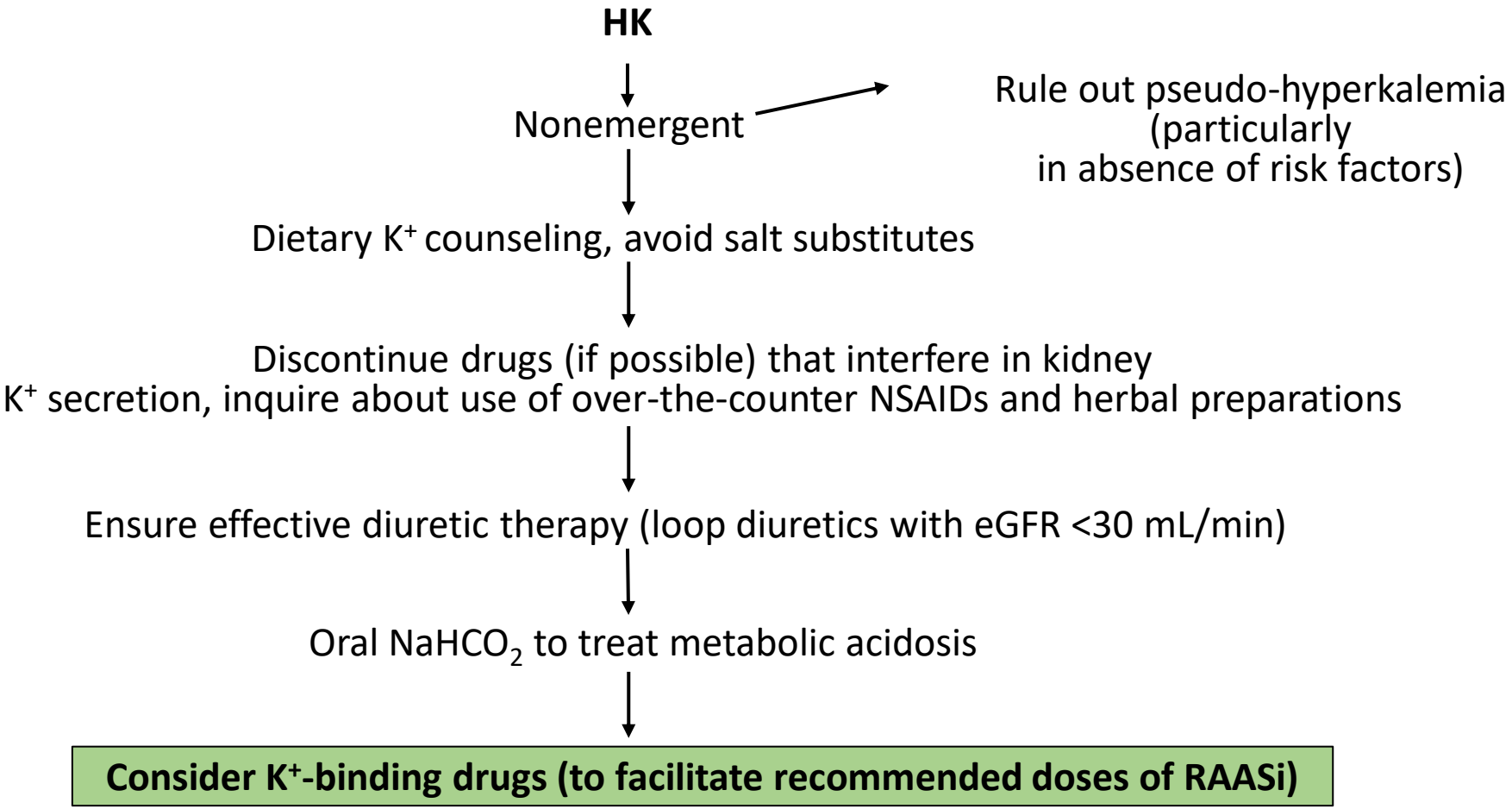
Emergent Treatment Algorithm



ESRD, end-stage renal disease; IV, intravenous.

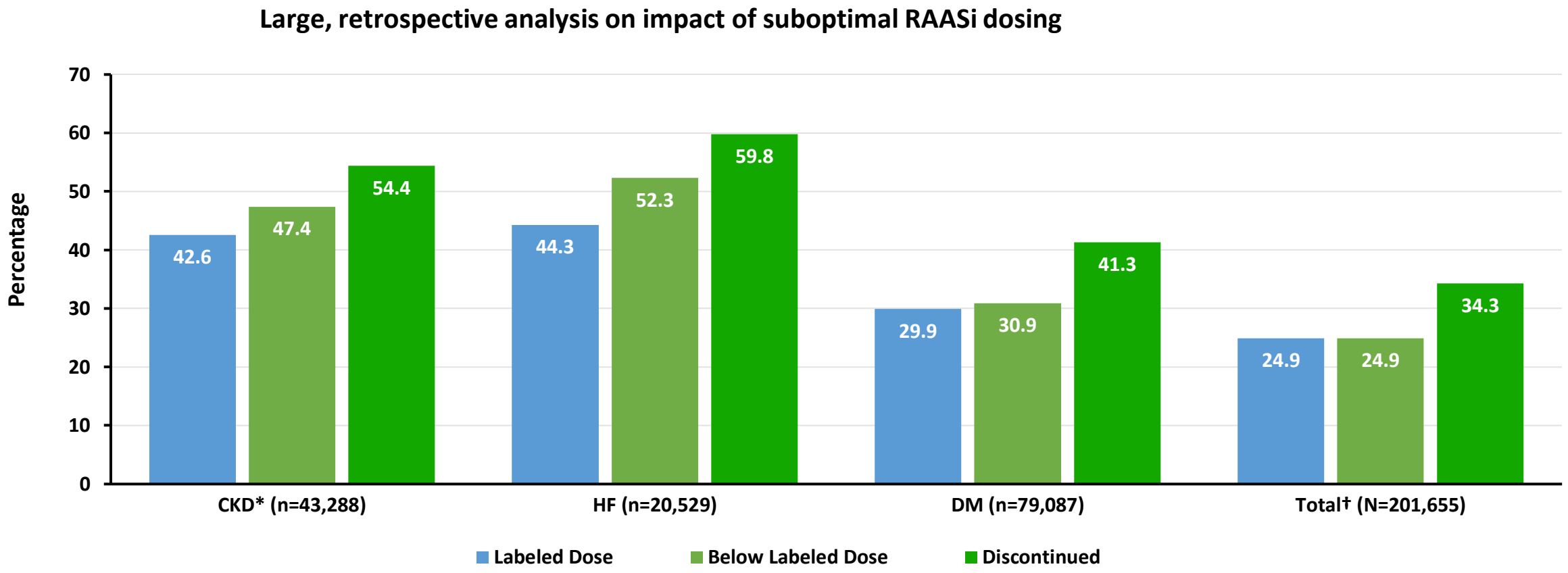
Adapted from: Palmer BF, Clegg DJ. *Am J Kidney Dis*. 2019;74:682-695.

Nonemergent Treatment Algorithm



Adapted from: Palmer BF, Clegg DJ. *Am J Kidney Dis*. 2019;74:682-695.

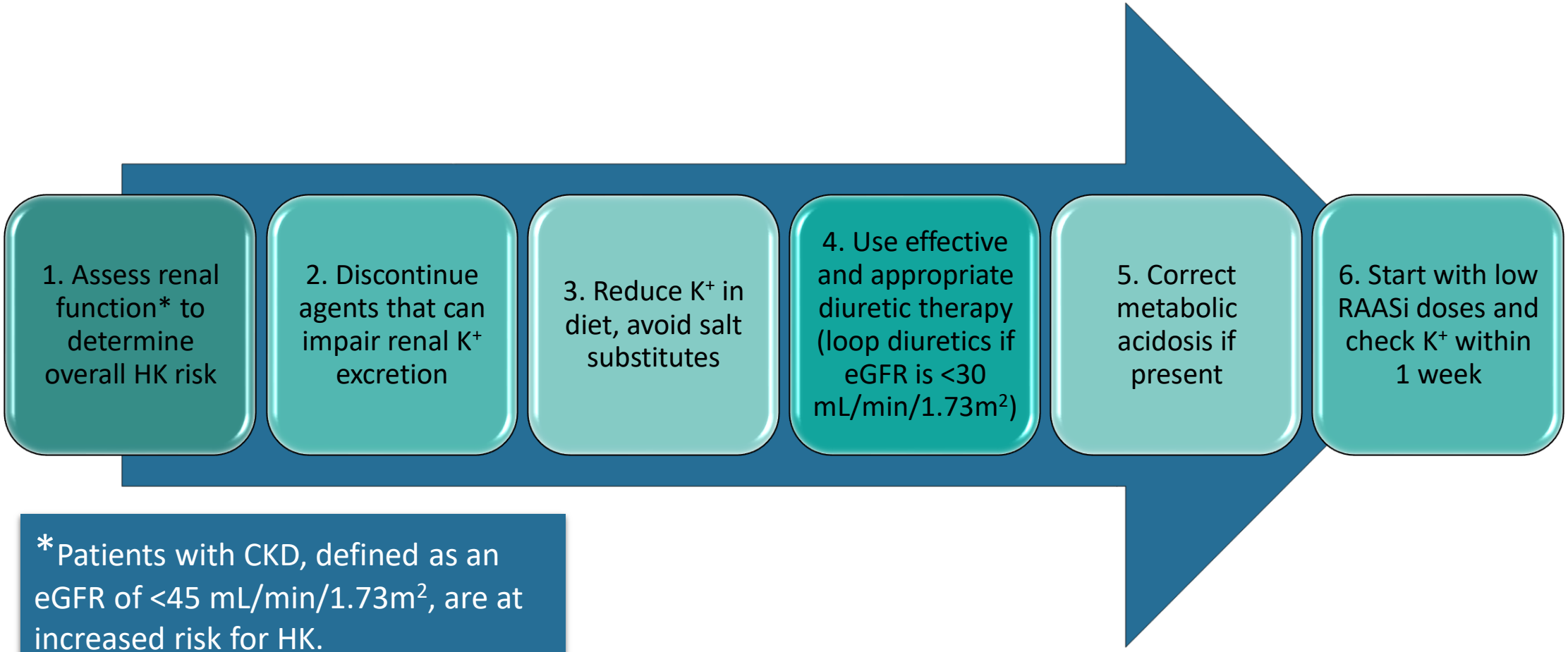
Reducing or Discontinuing RAASi Dose Linked to Poor Outcomes or Death



*Stages 3-4; lower stages and end-stage 5 disease not included. †Across all comorbidity and dose categories.

Adapted from: Epstein M, et al. *Am J Manag Care.* 2015;21(11 Suppl):S212-S220.

Steps to Reduce HK Risk When Using RAASi



*Patients with CKD, defined as an eGFR of <45 mL/min/1.73m^2, are at increased risk for HK.

Palmer BF, Clegg DJ. *Cleve Clin J Med.* 2017;84:934-942.

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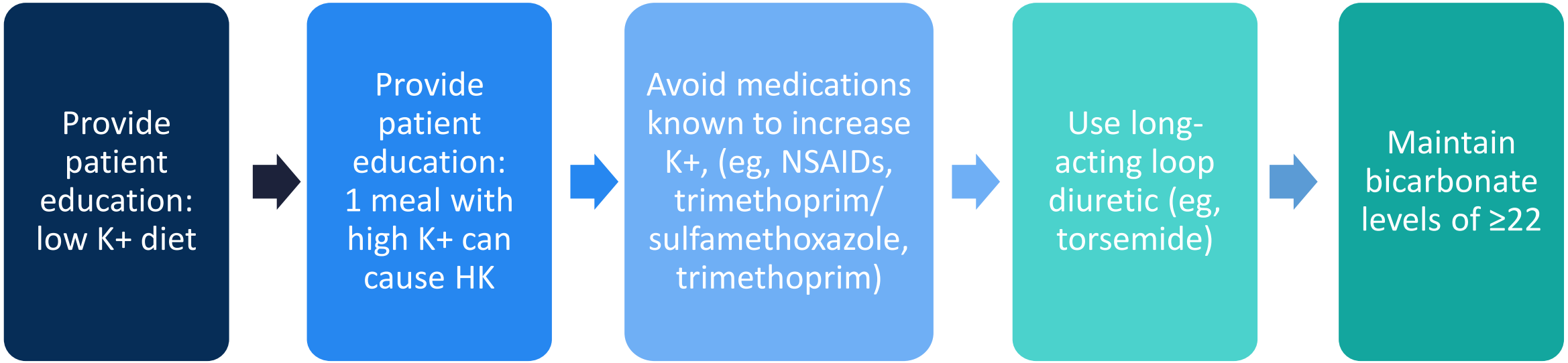
Evidence-Based Management of Hyperkalemia: The Role of Loop Diuretics

Approach to Avoid Acute HK in Patients With Known Chronic K+ Elevations



Palmer BF, et al. *Cleve Clin J Med*. 2017;84(12):934-942. doi:10.3949/ccjm.84a.17056; Wooten JM, et al. *South Med J*. 2019;112(4):228-233.doi:10.14423/SMJ.0000000000000957

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Evidence-Based Management of Hyperkalemia: The Role of Potassium-binding Agents in Treating Hyperkalemia

Sodium Polystyrene Sulfonate (SPS)

Sodium Zirconium Cyclosilicate (SZC)

Patiromer

Potassium Binders for Chronic HK

New K^+ binders can prevent HK episodes, while allowing RAASi to continue at optimal doses

These nonabsorbable agents are available in a powder formulation that can be mixed with water

They increase K^+ excretion through the GI tract

- Patiromer – exchanges K^+ for Ca^{2+} in the colon
- SZC – selectively binds K^+ ions and exchanges them for Na^+ and H^+ throughout the entire intestine

Patiromer (branded name) PI, 2020; Pitt B, Bakris GL. *Hypertension.* 2015;66:731-738; Sodium zirconium cyclosilicate (branded name) PI, 2020.

Potassium Binders: Top-level Comparisons

	SPS[1]	Patiromer[2]	SCZ[3]
Molecule[4]	Nonspecific, Na^+-containing organic resin	Selective, NA^+-free organic polymer	Selective, NA^+-containing inorganic crystalline silicate
Onset of effect[4]	Unknown*	7 hr	1 hr
Time to normokalemia[5]	Undetermined	79% after 4 wk	98% w/in 48 hr
Dosing[1-3]	Orally: 15–60 g, 1–4x/d; Rectally: 30–50 g/6 hr	8.4–25.2 g 1x/day	Starting: 10 g 3x/d up to 3 d†; Maintenance: 5 g, 10 g, 15 g 1x/d
Safety[4]	GI symptoms, hypokalemia, hypernatremia, volume overload	GI symptoms, hypokalemia, hypomagnesemia	Hypokalemia, edema

*Presume 7–10 hrs because SPS dependent on high concentration of K^+ in colon. †Package insert advises 3x/day up to 48 hrs; dosing based on ZS-005 trial: 82% of patients achieved normokalemia within 24 hrs.

1. Sodium polystyrene sulfonate (branded name) PI, 2017. **2**. Patiromer (branded name) PI, 2020. **3**. Sodium zirconium cyclosilicate (branded name) PI, 2020. **4**. Di Nicola L, et al. *J Nephrol.* 2018;31:653-664. **5**. Pitt B, Bakris GL. *Hypertension.* 2015;66:731-738

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New and Emerging Data in Hyperkalemia Management

Patiromer Efficacy and Safety Data

Sodium Zirconium Cyclosilicate (SZC) Efficacy and Safety Data

Patiromer: Phase 2 and 3 Trials

Study Name & Phase	Patient Population	Duration	Main Results
PEARL-HF[1] Phase 2	N=105 Chronic HF, CKD, or prior HK	4 wk	Significantly ↓ K^+ levels of –0.45 mEq/L vs placebo (*P*<0.001)
AMETHYST-DN[2] Phase 2	N=306 T2D w/ CKD and HK receiving RAASi	52 wk	Significant mean ↓ K^+ levels each month for entire 52 wk in pts w/ mild & moderate HK (*P*<0.001)
OPAL-HK[3] Phase 3	Phase A: N=219 Phase B: N=107 CKD w/ HK receiving RAASi	Phase A: 4 wk Phase B: 8 wk	Phase A: mean K^+ ↓ of –1.01 mEq/L (*P*<0.001); 76% reached target K^+ level by week 4 Phase B: 15% of pts taking patiromer had HK vs 60% taking placebo (*P*<0.001)

T2D, type 2 diabetes. Pitt B, Anker SD, Bushinsky DA, et al. *Eur Heart J*. 2011;32:820–828. Bakris GL, et al. *JAMA.* 2015;314:151-161. Weir MR, Bakris GL, et al. *N Engl J Med.* 2015;372:211-221.

Efficacy Studies of Patiromer in HK

Study		Baseline K^+, mmol/L	Number of Patients	Patiromer Dose	Change in Serum Potassium, mmol/L	*P* Value
AMETHYST-DN	Mild HK	5.1	74	8.4 g	–0.35	<0.001
		5.2	73	16.8 g	–0.51	<0.001
		5.1	73	25.2 g	–0.55	<0.001
	Moderate HK	5.7	26	16.8 g	–0.87	<0.001
		5.7	28	25.2 g	–0.97	<0.001
		5.6	30	33.6 g	–0.92	<0.001
OPAL-HK	**Initiation phase**					
	Mild HK	5.6	92	8.4 g	–0.65	<0.001
	Moderate-to-severe HK	4.49	151	16.8 g	–1.23	<0.001
	Randomized withdrawal phase					
		4.45	55	8.4 g or 16.8 g	0	<0.001
		5.9	52	Placebo	+0.72	<0.001

Wai H-T, et al. *Brit Jour of Card*. 2021; doi:10.5837/bjc.2021.014

Efficacy Studies of Patiromer in HK *(Cont)*

Study		Baseline K^+, mmol/L	Number of Patients	Patiromer Dose	Change in Serum Potassium, mmol/L	*P* Value
PEARL-HF	eGFR <60 mL/min		15	30g	–0.14	0.031
	eGFR >60 mL/min		40	30 g	–0.32	0.001
	History of HK		22	30 g	–0.34	0.058

Wai H-T, et al. *Brit Jour of Card*. 2021; doi:10.5837/bjc.2021.014

Summary of Patiromer AEs and Discontinuation Rate

Study	≥1 AE	Constipation	Diarrhea	Nausea/ Vomiting	Hypokalemia	Hypomagnesemia	Discontinued
PEARL-HF	54%	5%	5%	4%	6%	24%	7%
OPAL-HK	47%	11%	3%	3%	6%	3%	6%
AMETHYST-DN	69%	7.3%	5.6%	4%	5.6%	8.6%	9.2%

AE, adverse event.

Wai H-T, et al. *Brit Jour of Card*. 2021; doi:10.5837/bjc.2021.014

SZC: Phase 3 Trials

Study Name	Patient Population	Duration	Main Results
ZS-003[1]	N=753 DM, HF, CKD, w/ & w/o HK	14 d	↓ K^+ levels within 48 hr across all dosing groups; normokalemia maintained for 14 d (*P*<0.001) Onset of action observed within 1st hr
HARMONIZE[2]	Phase A: N=253 Phase B: N=237 DM, HF, CKD, with HK, most receiving RAASi	Phase A: 48 hr Phase B: 28 d	Phase A: ↓ K^+ levels within 48 hr; 98% achieved normokalemia; median time to normokalemia: 2.2 hr Phase B: Normokalemia of 80%–94% maintained across all dosing groups vs 46% taking placebo (*P*<0.001)
HARMONIZE Extension[3]	N=123 Open-label extension for HARMONIZE pts	≤11 mo	Normokalemia maintained for up to 11 mo No new safety signals emerged; patients able to remain on stable or increased RAASi doses

HTN, hypertension; pts, patients; wk, week.

1. Packham DK, et al. *N Engl J Med.* 2015;372:222-231. **2**. Kosiborod M, et al. *JAMA.* 2014;312:2223-2233.
3. Roger SD, et al. *Am J Nephrol.* 2019;50:473-480.

SZC: Phase 3 Trials *(Cont)*

Study Name	Patient Population	Duration	Main Results
ZS-005[1]	Phase A: N=751 Phase B: N=746 DM, HF, CKD, w/HK, HTN, & other comorbidities; most receiving RAASi	Phase A: up to 72 hr Phase B: 12 mo	Phase A: 99% achieved normokalemia (82% w/in 24 hr; 13% in 48 hr; 4% in 72 hr) Phase B: Serum K^+ ≤5.1 mEq/L reached by 88%, ≤5.5 by 99%; 74% of pts taking RAASi were able to maintain start dose
DIALIZE[2]	N=196 ESRD on hemodialysis 3x/wk with HK	8 wks	41.2% of the SZC cohort met the primary endpoint vs 1% of those randomized to placebo

1. Spinowitz BS, et al. *Clin J Am Soc.* 2019;14:798-809**. 2**. Fishbane S, et al. *J Am Soc Nephrol.* 2019;30:1723-1733.

Efficacy of SZC in HARMONIZE

Study	Length, months	Baseline K^+, mmol/L	No. of Patients	Dose		Mean Serum Potassium, mmol/L	
				Correction Phase	Maintenance	≤5.1 mmol/L	≤5.5 mmol/L
HARMONIZE extension	11	4.8	123	10 g tds 3–6 doses	<10 g qds 13.8% 10 g qds 73.2% >10 g qds 13.0%	88.3% 95% CI 81.2% to 93.5% *P*<0.001	100.0% 95% CI 97.0% to 100.0% *P*<0.001
12-month phase III	12	5.6	751	10 g tds 3–6 doses	5 g od 47% 10 g od 41% 15 g od 12%	88.0%	99.0%

od, once daily; qds, four times daily; tds, three times daily.

Wai H-T, et al. *Brit Jour of Card*. 2021; doi:10.5837/bjc.2021.014

SZC Overall Safety

Not studied in patients with severe constipation, bowel obstruction or impaction, including abnormal post-operative bowel motility disorders, and may be ineffective and may worsen gastrointestinal conditions.

Edema: In patients who were not on dialysis, edema was observed and was generally mild to moderate and more commonly seen in patients treated with 15 g once daily.

Patients on chronic hemodialysis in which most patients were treated with doses of 5 g to 10 g once daily on nondialysis days, there was no difference in the mean change from baseline in interdialytic weight gain between SZC and placebo groups.

HK in Patients on Hemodialysis: Patients on hemodialysis may be prone to acute illness that can increase the risk of HK on SZC (eg, illnesses associated with decreased oral intake, diarrhea).

AEs: The most common AE in non-dialysis patients was mild to moderate edema. In placebo-controlled trials up to 28 days, edema was reported in 4.4%, 5.9%, 16.1% of nondialysis patients treated with 5 g, 10 g, and 15 g of SZC once daily, respectively, vs 2.4% of nondialysis patients receiving placebo.

https://medicalinformation.astrazeneca-us.com/

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Emerging Agents With Potential Benefit to Hyperkalemia

DAPA-HF (Dapagliflozin And Prevention of Adverse Outcomes in Heart Failure) Trial

DAPA-HF: A Secondary Analysis

Dapagliflozin (SGLT2 inhibitor) reduces the risk of hyperkalemia in patients with heart failure and reduced ejection fraction

3370 (70.1%) patients (65 vs 69 yo) treated with MRA

Mild hyperkalemia (serum K+ >5.5 mmol/L) and moderate-to-severe hyperkalemia (> 6.0 mmol/L) occurred in 182 (11.1%) and 23 (1.4%) patients treated with dapagliflozin vs 204 (12.6%) and 40 (2.4%) of placebo patients

Dapagliflozin reduced risk for mild hyperkalemia by 14% and moderate-to-severe hyperkalemia by 50% (*P*=.01) vs placebo

Moderate-to-severe hyperkalemia was reduced from 1.7 to 1.0 per 100 person-years with dapagliflozin

SGLT2, sodium-glucose cotransporter.

Kristensen SL, et al. Presented at ESC Virtual Congress 2020, August 29, 2020.

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Patient Case Scenario:
Patient With Heart Failure and Possible Hyperkalemia

Patient Case: Background

Background

George is a 64yo man with HF who is taking an ACE inhibitor. He presents to his clinician with generalized edema, weight gain, shortness of breath, and occasional heart racing/palpitations.

Discussion

- What additional questions would you ask?
- What tests would you request?
- Would you admit him to the hospital?

Sidhu K, et al. *Curr Op in Card.* 2020;35(2):150-155. doi: 10.1097/HCO.0000000000000709

Patient Case: Polling Question 1

When evaluating George, which factor(s) will indicate risk of HK?

A. eGFR <35 mL/min/1.73 m^2
B. Use of RAASi, NSAIDs, or antibiotics
C. K+ >4.8.mEq/L without RAASi
D. A & C
E. All of the above

Patient Case: Evaluation and Assessment

Evaluation

- Medical history: HF with reduced left ventricular ejection fraction 31.4%
- Examination: systolic blood pressure 120.3
- Labs: (K+ >6.0 mmol/L); eGFR 30 ml/min/1.73 m^2
- Medications: RAASi; specifically, ACE inhibitor

Assessment

The patient's kidney function has deteriorated and labs confirm HK

Discussion

- How would you manage George?
- Which treatment(s) would you choose and at what dose(s)?

Sidhu K, et al. 2020. *Curr Op in Card.* 35:2:150-155 doi: 10.1097/HCO.0000000000000709

Patient Case: Polling Question 2

George has been diagnosed with HK. What is the next step to reduce his HK events for optimal outcomes?

A. Discontinue ACE inhibitor and prescribe a resin binder
B. Reduce the ACE inhibitor dose and request patient to be tested weekly for K^+ serum concentration levels
C. Prescribe a K^+ binder to allow continued optimal ACE dosing regimen
D. Prescribe RAAS inhibitor with a mechanism of action different than that of the ACE inhibitor (eg, ARB)

Patient Case Study: Patiromer & SZC Are Useful Adjuncts in Clinical Care of patients With HF & HK

Therapeutic Management

George may be prescribed SCZ or patiromer to manage HK, while allowing him to maintain HF treatment with ACE inhibitor

- Both agents studied in HF and demonstrated maintained normokalemia for extended periods of time with improved side effect profiles vs existing K+ binders
- No direct comparisons have occurred
- SZC has also shown promise in treatment of acute HK with quick onset of action
- Both agents may allow clinicians to maintain patients on RAASi and uptitrate guideline directed medical therapy to target doses without concern for recurrent HK

Discussion

- How would you instruct George?
- What would you prescribe at discharge?

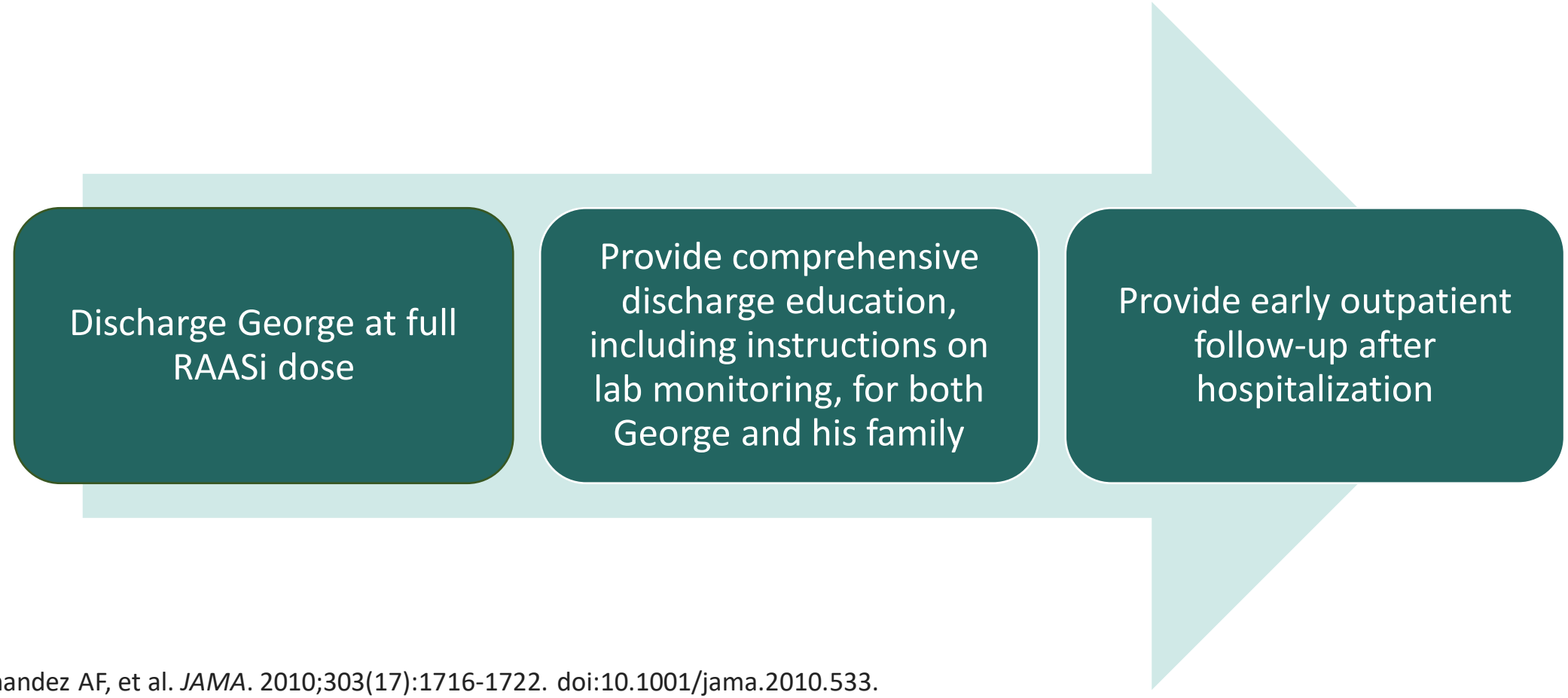
Sidhu K, et al. *Curr Op in Card.* 2020;35:2:150-155. doi: 10.1097/HCO.0000000000000709

Patient Case: Polling Question 3

To reduce the risk of readmission for George, what steps should be taken when he is discharged?

A. Focus discharge instructions on prescribed medications and basic therapeutic regimen
B. Provide comprehensive discharge education, including instructions on lab monitoring, to patient and family
C. Ensure patient commitment to follow-up after hospitalization if signs or symptoms emerge
D. Be sure patient understands when to seek treatment, rather than what HK is and relation to K+

Patient Case Study: Discharge and Follow Up



Hernandez AF, et al. *JAMA*. 2010;303(17):1716-1722. doi:10.1001/jama.2010.533.
National Kidney Foundation. Accessed Oct. 8, 2021. https://www.kidney.org/sites/default/files/02-10-7271_ABG_Hyperkalemia_Card_P11b.pdf

Optimizing Multidisciplinary Care and Transitions From Hospital to Home

Key Strategies for Improved Outcomes

In-hospital, multidisciplinary rapid-response teams can facilitate diagnosis and management of HK

- Improve patient outcomes
- Enhances treatment efficacy
- Optimize HK recognition in high-risk patients
- Quickly identify patients who are not truly hyperkalemic and don't require treatment

Reduce readmission for patients with chronic disease who may be at risk for HK:

- Provide early outpatient follow-up after hospitalization
- Provide discharge education for patients/family
- Multidisciplinary team members are in an important position to provide patients with comprehensive education:
  - Condition/disease state
  - Medications
  - When to seek medical treatment

What HK is and its link to K+

Why patient is at risk for HK

Symptoms to watch out for

- Unexplained fatigue, muscle weakness, heart palpitations

What to avoid to reduce HK risk

- Foods high in potassium ***if*** the patient has kidney disease (most such foods are very good for heart health and should be encouraged otherwise)
- Potassium supplements and salt substitutes that use potassium instead of sodium

Why it is important to take all medications as prescribed

National Kidney Foundation. https://www.kidney.org/sites/default/files/02-10-7271_ABG_Hyperkalemia_Card_P11b.pdf

Unmet Need for Inpatient and Post-discharge Management of HK Patients

Study Design and Patient Population

Retrospective cohort study using a large US claims database

Hyperkalemia cohort
Patients with hyperkalemia-related hospitalization

VS

Nonhyperkalemia cohort
Patients without evidence of hyperkalemia

Cohorts were matched 1:1 on age, CKD stage, heart failure, dialysis treatment, RAASi use, and major diagnostic categories of the hospitalization (N = 4426 pairs)

Results

Post-discharge outcomes (1-year)	Hyperkalemia cohort	Non-hyperkalemia cohort
Total costs	$68,861	$38,482
IP admissions	1.0	0.4
ED visits	2.0	1.2
OP visits	49.6	39.1
30-day readmissions	0.15	0.09
Total length of stay (days)	10.5	5.8

Note: all *P* values <.001

Results remained robust in multivariable regressions adjusting for additional comorbidities, characteristics of the hospitalization, and medication use

Conclusion: HK-related hospitalizations were associated with significant postdischarge burden. This suggests an unmet need for the inpatient and postdischarge management of hyperkalemia patients.

IP, inpatient; ED, emergency department; OP, outpatient.

Betts KA, et al. *Kidney Int Rep*. 2020;5(8):1280-1290. doi:10.1016/j.ekir.2020.06.004

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Summary & Closing

Program Summary

HK

- Common and potentially life-threatening electrolyte imbalance
- Most frequently seen in patients with advanced CKD and HF
- Can be missed as patients are often asymptomatic
- Potassium homeostasis a delicate balance within a very narrow range

HK and RAASi

- RAASi is one of many potential causes
- Discontinuing RAASi treatment may not be the right answer; multiple studies show worse outcomes when RAASi is discontinued or reduced, especially in patients with HF and CKD
- Instead, mitigate against HK and optimize RAASi using preventive approaches and K+ binders
- Diagnose HK quickly and monitor K+ levels frequently
- Discharge patients at full RAASi dose and teach patients how to avoid HK

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Thank you!

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