



# Potassium Management

ACC  MED

SEZIONE DI NEFROLOGIA

KDIGO

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con il Patrocinio



CONVEGNO » 4,8 CREDITI FORMATIVI

## GRANDANGOLO 2020 IN NEFROLOGIA, DIALISI E TRAPIANTO

SELEZIONE E ANALISI RAGIONATA  
DEI PIÙ RECENTI DATI SCIENTIFICI

XIV EDIZIONE

BOLOGNA, 6-7-8 FEBBRAIO 2020

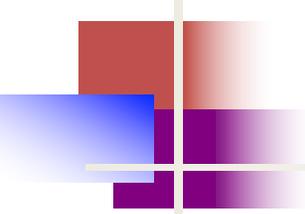
RESPONSABILI SCIENTIFICI

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# Disclosure of Interests

...received commercial/research assistance, and financial sponsorship to attend conferences, advisory boards and clinical trials from Vifor Pharma and AstraZeneca, and several “CKD-MBD treatment” companies





# Today's talk.....

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- Treatment options
  - What about the foods?
  - Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia
  - What is on the horizon to lower potassium?
- Unmet need of hyperkalemia in CKD: How would you manage the patient?

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RAASi = Renin Angiotensin Aldosterone System inhibitors  
MRA = Mineralocorticoid Receptor Antagonists

# Potassium homeostasis and management of dyskalemia in kidney diseases: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference

OPEN

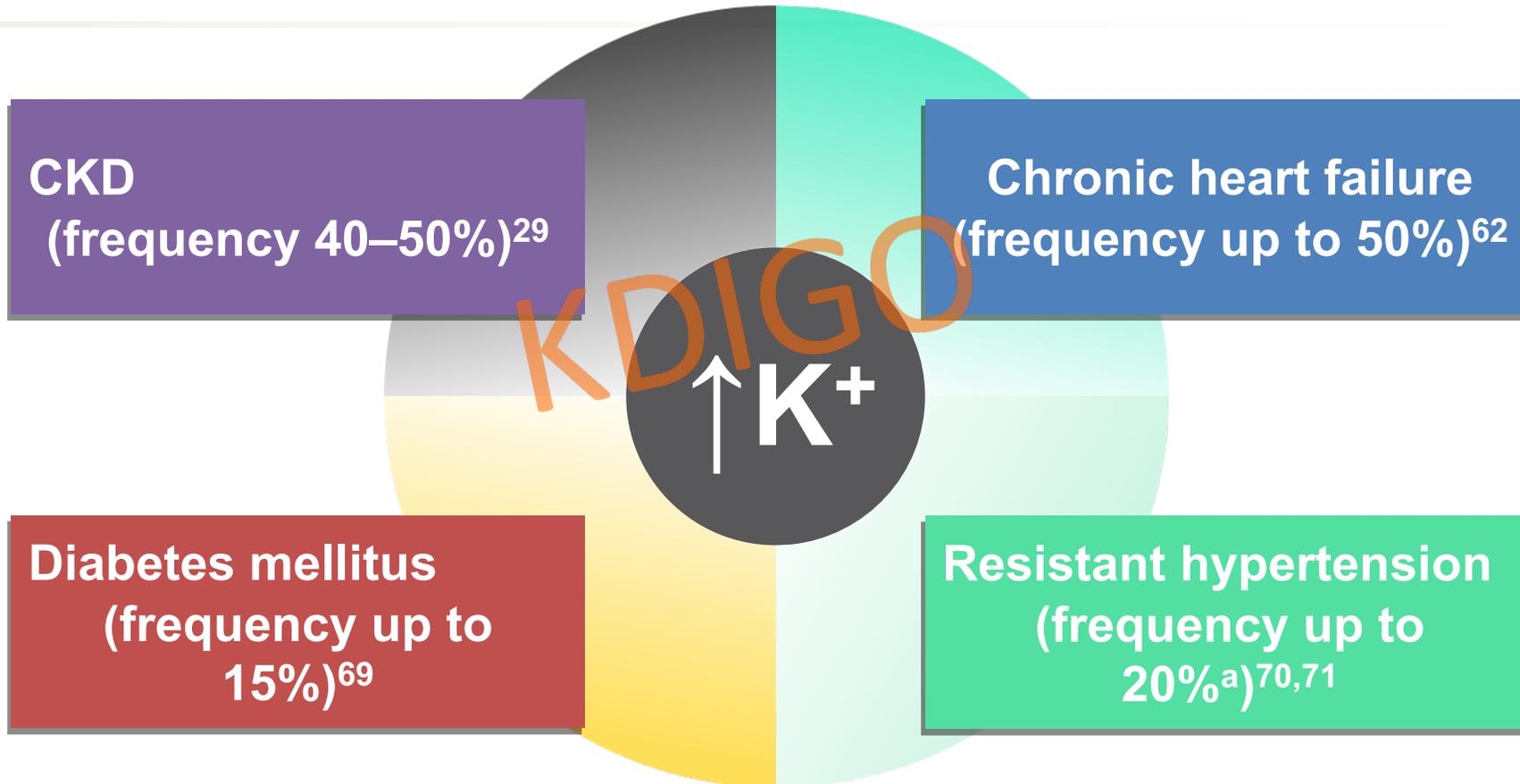
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KDIGO

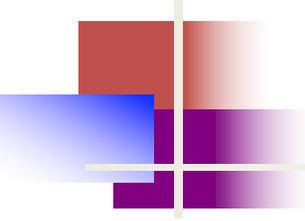
**A** multidisciplinary group of researchers and clinicians met in October 2018 to identify evidence and address controversies in potassium management. Here we provide our overview of potassium homeostasis in health and disease and guidance for evaluation and management of dyskalemias in the context of kidney diseases, and indicate research priorities.



***Hyperkalaemia is prevalent in specific patient populations, such as CKD and heart failure and those prescribed key drug classes***



<sup>a</sup>Among patients prescribed add-on MRA treatment  
CKD, chronic kidney disease; HF, heart failure; MRA, mineralocorticoid receptor antagonist



# Today's talk.....

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- Treatment options

- **What about the foods?**

- Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia

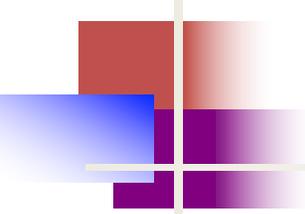
- What is on the horizon to lower potassium?

- Unmet need of hyperkalemia in CKD: How would you manage the patient?



RAASi = Renin Angiotensin Aldosterone System inhibitors

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# Dietary measures

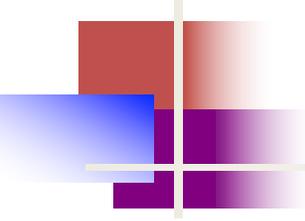
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- Restrict their intake of high-potassium foods (>250 mg (6mmol) per 100 g)
- Maintain a low-potassium diet (potassium intake of  $\leq 3$  g per day)

# Which food has the highest potassium content per usual serve?

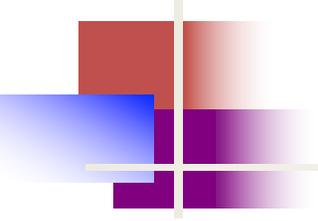
- 1) Banana
- 2) Tomato (3 slices)
- 3) Hot chips (small)
- 4) Iced-coffee (250mL)
- 5) Beans (1/2 cup)





**Potassium:**  
**1mmol = 39mg**

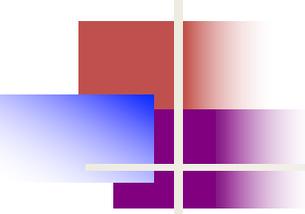
	<i>mmol potassium</i>
<b>Banana</b>	<b>9</b>
<b>Mandarin</b>	<b>3</b>
<b>Orange</b>	<b>5</b>
<b>Tomato (3 slices)</b>	<b>3</b>
<b>Mango (1 cheek)</b>	<b>5</b>
<b>Nectarine</b>	<b>9</b>
<b>Peach</b>	<b>7</b>
<b>Grapes</b>	<b>6</b>
<b>Mashed potato</b>	<b>10</b>
<b>Hot chips (small)</b>	<b>12</b>
<b>Juice (250mL)</b>	<b>10</b>
<b>Milkshake (250mL)</b>	<b>10</b>
<b>Ice-coffee (250mL)</b>	<b>10</b>
<b>Crisps (small packet)</b>	<b>15</b>
<b>Apple</b>	<b>4</b>
<b>Pear</b>	<b>4</b>
<b>Strawberries</b>	<b>5</b>
<b>Watermelon</b>	<b>6</b>
<b>Carrot (1/2 cup)</b>	<b>4</b>
<b>Beans (1/2 cup)</b>	<b>1</b>
<b>Zucchini (1/2 cup)</b>	<b>4</b>



# Lowering Potassium Levels

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- It's not all about bananas...
- Anyone can lower a patient's potassium, the dietitian's role, is to assess a patient's diet and ***negotiate changes*** while:
  - meeting patients preferences,
  - keeping the diet healthy
  - maintaining safe potassium levels
- The hospital low potassium diets are very limiting



# What is the normal potassium intake?

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1. 30-50mmol/d

2. 50-75mmol/d

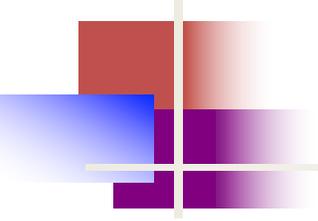
3. 75-100mmol/d

4. 100-150mmol/d

5. Don't know!

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# What is the normal potassium intake?

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75-100mol/L

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How do you assess potassium intake?



**Table 1 | Studies associating potassium intake, CKD outcomes, and mortality**

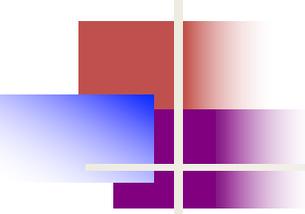
Study	Population	Dietary K <sup>+</sup> assessment	Outcome definitions	Factors associated with higher K <sup>+</sup> intake
Araki <i>et al.</i> , 2015 <sup>82</sup>	623 Japanese patients with diabetes and eGFR $\geq 60$ ml/min per 1.73 m <sup>2</sup> enrolled between 1996–2003 and followed up until 2013	Estimated from a single baseline 24-h urine collection	eGFR $\downarrow \geq 50\%$ or progression to CKD G4 or annual rate of eGFR decline	$\downarrow$ risk of both outcomes Slower rate of annual eGFR decline
Smyth <i>et al.</i> , 2014 <sup>71</sup>	Post hoc analysis of ONTARGET and TRANSCEND studies; >30,000 patients from 18 countries with vascular disease or diabetes with end-organ damage	Estimated 24-h urine K <sup>+</sup> from a single urine sample	eGFR $\downarrow \geq 30\%$ or CD, or eGFR $\downarrow \geq 40\%$ or CD, or rapid progression, or doubling of SCr or CD, or progression of proteinuria	$\downarrow$ risk of CKD progression
Kieneker <i>et al.</i> , 2016 <sup>85</sup>	5315 Dutch participants aged 28 to 75 yr in the PREVEND study and followed up for a median of 10.3 yr	Two 24-h urine collections at baseline and midway during follow-up	CKD incidence	$\downarrow$ risk of incident CKD
Smyth <i>et al.</i> , 2016 <sup>88</sup>	544,635 participants in the NIH-AARP Diet and Health Study, aged 51–70 yr	FFQ to assess K <sup>+</sup> intake over the preceding year	Death due to renal causes or need for dialysis	$\downarrow$ risk of both kidney outcomes
Leonberg-Yoo <i>et al.</i> , 2017 <sup>86</sup>	Post hoc analysis of MDRD study; 812 patients aged 15–70 yr with CKD G2–G4	Estimated from 24-h urine collection at baseline and at multiple time points	Initiation of chronic dialysis or kidney transplantation (kidney replacement therapy) Death from all causes	No association with kidney replacement therapy Association with $\downarrow$ risk of death
Mirmiran <i>et al.</i> , 2018 <sup>87</sup>	1780 participants in the Tehran Lipid and Glucose study and followed up for 6.3 yr	Validated 168-item FFQ	CKD incidence	No association
He <i>et al.</i> , 2016 <sup>84</sup>	3939 participants aged 21–74 yr with CKD (GFR 20–70 ml/min per 1.73 m <sup>2</sup> ) in the CRIC study	Estimated from 24-h urine collection at baseline and at years 1 and 2	Composite of ESKD or halving of GFR Death from all causes	$\uparrow$ risk of CKD progression No association with risk of death
Noori <i>et al.</i> , 2010 <sup>69</sup>	224 chronic HD patients from the NIED Study	Estimated 24-h urine K <sup>+</sup> from FFQ	Death from all causes	$\uparrow$ risk of death only when comparing extreme intakes
Eisenga <i>et al.</i> , 2016 <sup>83</sup>	Prospective cohort of 705 stable kidney transplant recipients	A single 24-h urine collection and FFQ	Graft failure Death from all causes	$\downarrow$ risk of graft failure and death
Kim <i>et al.</i> , 2019 <sup>89</sup>	1821 participants aged 20–75 yr with CKD G1–G5 (nondialysis) in the KNOW-CKD study	24-hour urine collection at baseline; spot urine	Composite of GFR $\downarrow \geq 50\%$ or ESKD	$\downarrow$ risk of CKD progression

Direct evidence in support of the current recommendation for restricting dietary potassium in patients with CKD was lacking; however, we did not find evidence that increased potassium intake, or liberalization of potassium restrictions, in patients with advanced CKD is safe. While we acknowledge that dietary potassium restriction is a valid strategy to treat acute hyperkalemia, we hypothesize that potassium restriction as a general strategy to prevent hyperkalemia in persons with CKD may deprive patients of the beneficial effects associated with potassium-rich diets. We recommend that interventional trials be conducted to clarify optimal dietary potassium advice for patients with

**Table 2 | Summary of evidence and future research recommendations for dietary potassium in CKD**

What we know	What we think	Future research
<p>K<sup>+</sup>-rich diets are consistent with fruit and vegetable-rich healthy dietary patterns.</p> <p>K<sup>+</sup> supplementation, at a general population level, reduces blood pressure and lowers the risk of stroke.</p> <p>In people with CKD, estimations of dietary K<sup>+</sup> correlate poorly with circulating K<sup>+</sup>.</p>	<p>Generalized dietary K<sup>+</sup> restriction in people with CKD may deprive them from other beneficial effects and nutrients of K<sup>+</sup>-rich diets.</p>	<ul style="list-style-type: none"><li>• Investigate the effect of dietary K<sup>+</sup> restriction in CKD on circulating levels</li><li>• Investigate the effect of fruit- and vegetable-rich diets in CKD</li><li>• Develop new methods and validate existing methods to estimate dietary K<sup>+</sup> intake in people with CKD</li><li>• Evaluate the impact of dietary K<sup>+</sup> on serum concentration in people with CKD</li><li>• Evaluate the effects of dietary K<sup>+</sup> restriction in people with CKD on clinically important outcomes, including harms</li><li>• Evaluate the effects of unrestricted fruit/vegetable intake on the risk of hyperkalemia in people with advanced CKD or who are undergoing dialysis</li></ul>

CKD, chronic kidney disease; K<sup>+</sup>, potassium.

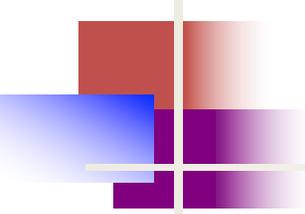


# Today's talk.....

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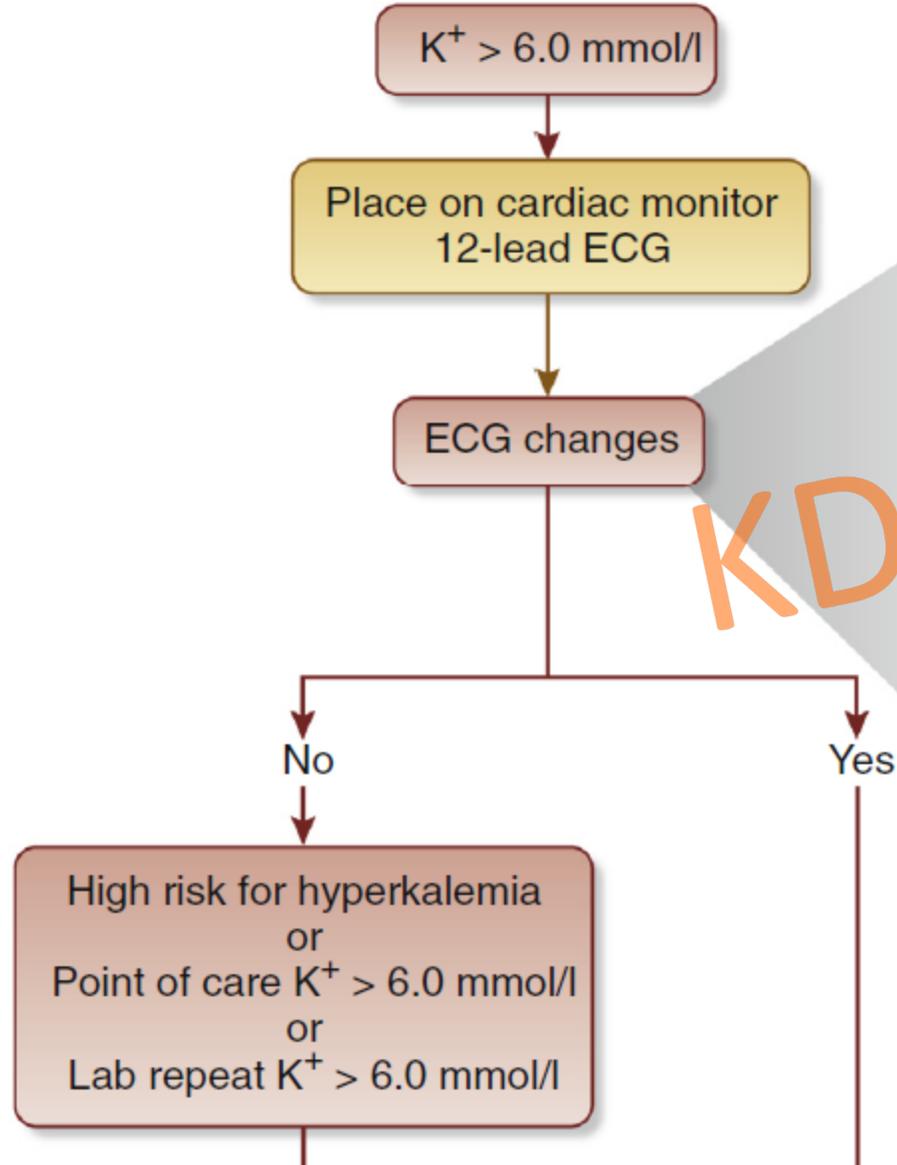
- Treatment options
  - What about the foods?
    - **Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia**
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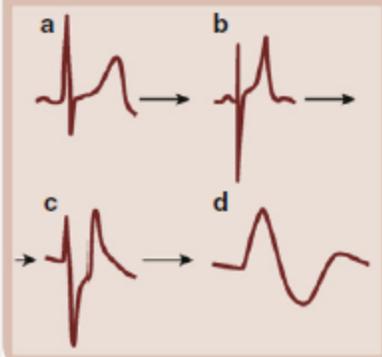


ECG changes	+	Moderate	Severe	Severe
	-	Mild	Moderate	Severe
		5.0*–5.9	6.0–6.4	≥6.5
		Potassium concentration (mmol/l)		

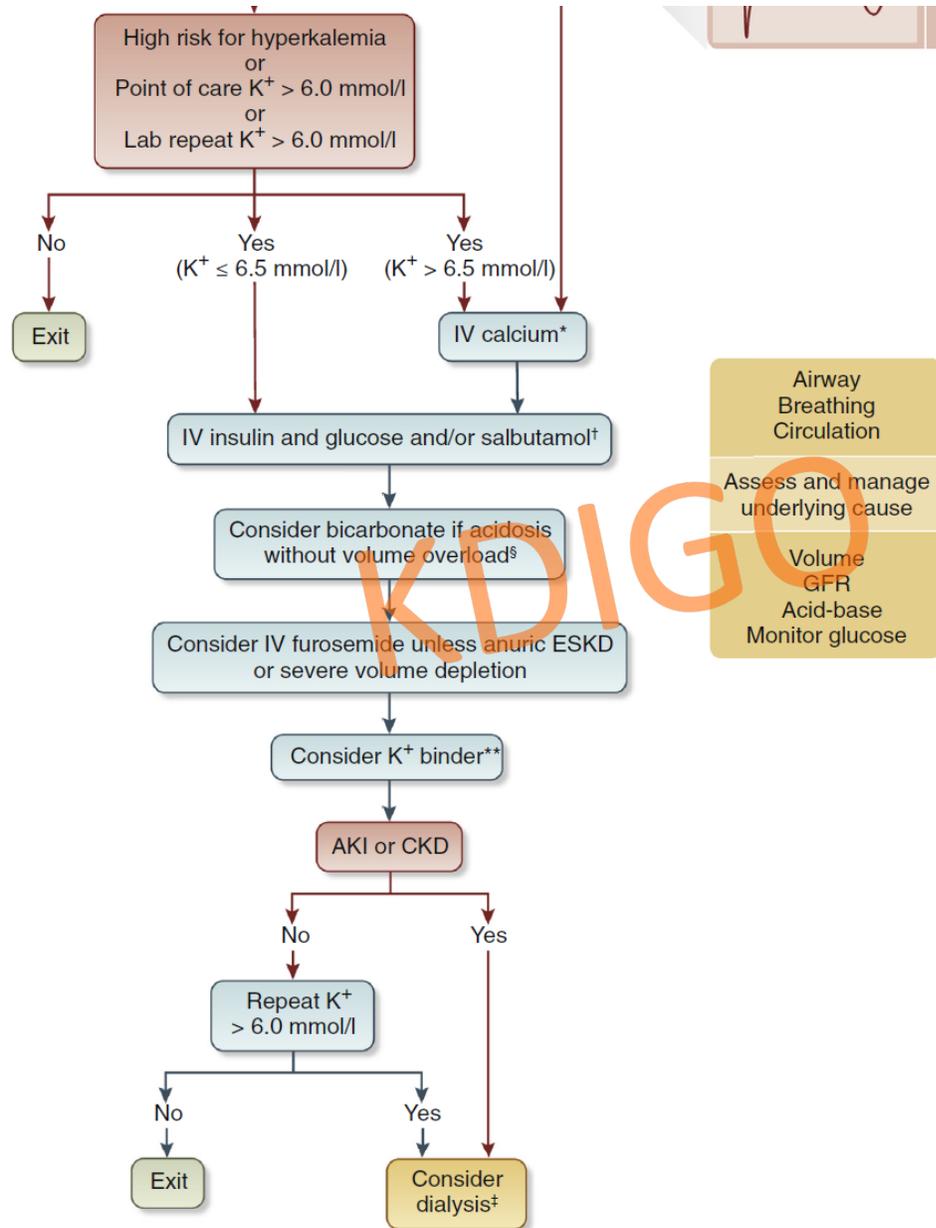
**Figure 4 | Severity of acute hyperkalemia: expert opinion-based risk classification.** \*5.0 or upper limit of normal range. ECG, electrocardiogram.



Serum potassium	Expected ECG abnormality
5.5–6.5 mmol/l	Tall, “peaked” T waves with narrow base, best seen in precordial leads
6.5–8.0 mmol/l	Peaked T waves Prolonged PR interval Decrease amplitude of P waves Widening of QRS complex
>8.0 mmol/l	Absence of T wave Intraventricular blocks, fascicular blocks, bundle branch blocks, QRS axis shift Progressive widening of QRS resulting in bizarre morphology “Sine wave” patterns (sinoventricular rhythm), VF, asystole



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## Table 5 | Future questions for hyperkalemia research

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### Acute hyperkalemia

Understanding the burden of disease

Testing the efficacy of acute management

Understanding the measurement properties of ECG  
in the prediction of clinical outcomes

Testing the efficacy of noninvasive screening for  
hyperkalemia

- Determine the prevalence of hyperkalemia in patients with acute kidney injury
- Compare the efficacy and safety of calcium chloride and calcium gluconate for management of acute hyperkalemia with ECG changes
- Evaluate the efficacy of loop diuretics for treatment of acute hyperkalemia
- Determine the ECG changes that warrant administration of intravenous calcium salts
- Evaluate use of artificial intelligence and smart phone technology for noninvasive monitoring to detect hyperkalemia in the outpatient setting

## Chronic hyperkalemia

Testing the efficacy and harms of dietary intervention

Testing the efficacy of newer agents for potassium reduction in populations not well represented in current trials

Evaluating the risks and benefits of maintaining and optimizing RAAS blockade despite hyperkalemia

Preventing clinical events arising from hyperkalemia

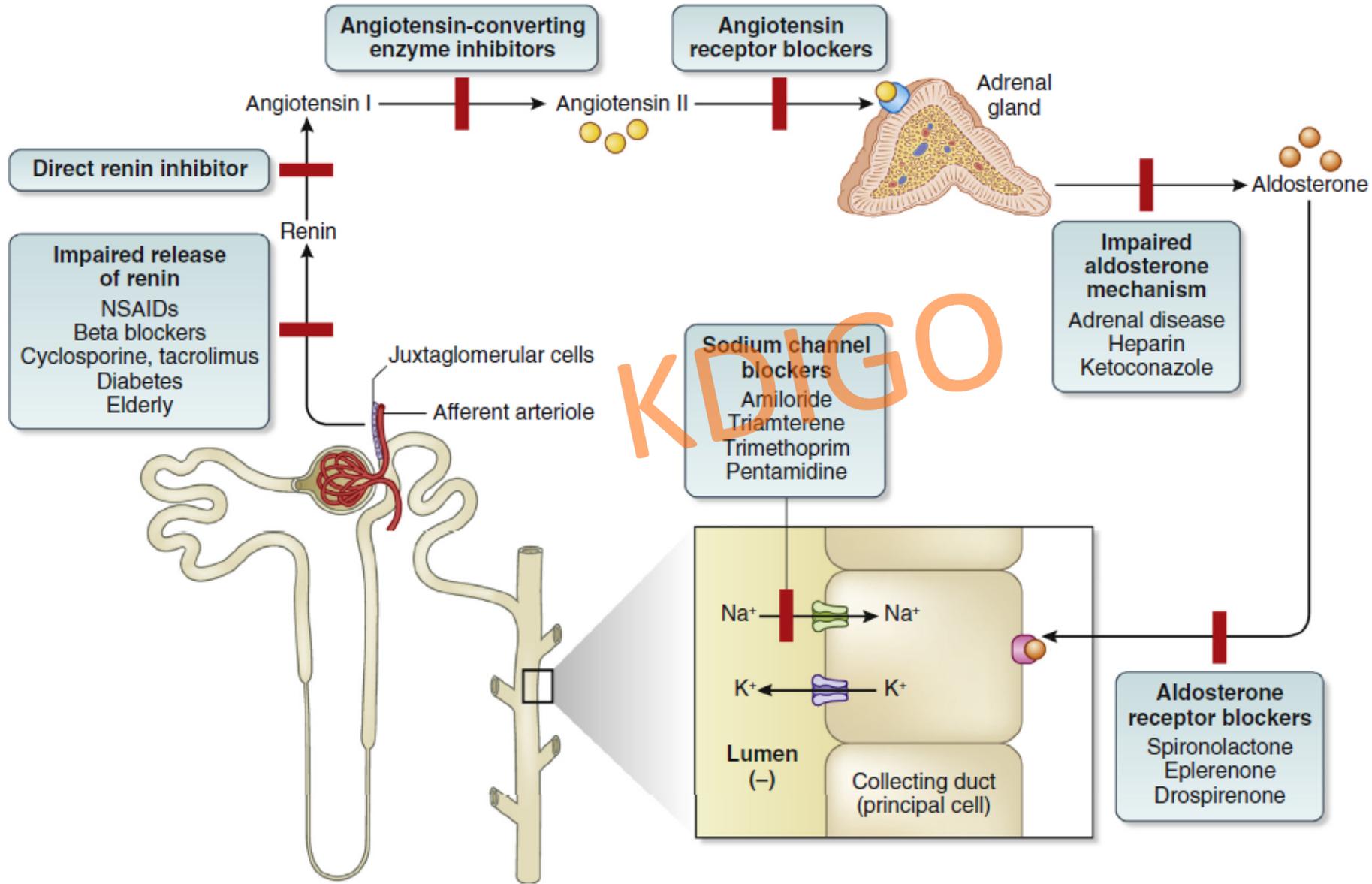
Preventing health service utilization arising from hyperkalemia

Testing the impact of the newer agents on the patient experience using patient reported outcomes

Testing the relative role of the newer agents

Understanding the dynamics of potassium

- Including PROs and clinically important cardiorenal outcomes, as well as serum potassium concentration
- In ESKD people receiving maintenance hemodialysis
- In people with functioning kidney transplants, test efficacy and harms, investigate impact on immunosuppressant levels
- In diverse populations (differing by clinical context, ethnicity, or diet)
- In people with type 4 RTA and normal or reduced eGFR
- In people with heart failure for cardiovascular outcomes
- In people at risk of progression for preventing CKD progression
- In people with hyperkalemia (e.g., diabetes with type 4 RTA, advanced CKD, ESKD receiving hemodialysis) for survival and prevention of arrhythmias
- Reduction of hospitalizations, emergency department presentations, investigations, and management arising as a response to hyperkalemia
- PROs for standard of care vs. newer strategies
- Gut symptoms, pill burden, illness intrusiveness (medication timing away from binders, monitoring, possible diet liberalization), peripheral neuropathy<sup>99</sup>
- Important to compare with placebo and with SPS (lower cost; wide experience)
- In broad populations head-to-head trials, including comparison with SPS (lower cost; wide experience)
- Directly assessing and modeling the impact of ability to continue RAAS blockade
- Monitoring frequency; novel continuous monitoring devices may provide data for modeling studies to define optimal monitoring frequencies
- Deprescribing trials



**Table 6 | Approaches to the management of chronic hyperkalemia**

Strategy	Comment
Dietary potassium restriction	<ul style="list-style-type: none"><li>• Reliant on lifestyle change</li><li>• Uncertainty on degree and reliability of response</li><li>• Poor evidence base to support the practice</li><li>• Financial cost of special diets</li><li>• Practical issues in implementation</li><li>• Potential for harm because of impact of diet on intake of other beneficial nutrients, healthy dietary pattern</li><li>• Potential for harm through loss of enjoyment in food and impact on social activities</li></ul>
Permissive approach (no additions or changes to management despite awareness of hyperkalemia)	<ul style="list-style-type: none"><li>• The extent of practice poorly documented</li><li>• Potentially could be tested in randomized trials given the uncertainty on benefits and harms of approaches based on tolerance of different potassium thresholds</li></ul>
Discontinuation of medications elevating potassium (e.g., RAAS inhibitors)	<ul style="list-style-type: none"><li>• Common strategy</li><li>• Effect on outcomes unknown<sup>168,204</sup></li></ul>
Use of potassium-wasting diuretics	<ul style="list-style-type: none"><li>• Dependent on kidney function; RCT evidence of no impact on potassium concentrations in people on PD with residual kidney function<sup>213</sup>; small pre-post studies suggest that metolazone but not thiazides may be kaliuretic in patients with GFR &lt;20 ml/min per 1.73 m<sup>2</sup><sup>214,215</sup></li><li>• Degree and predictability of response uncertain</li><li>• Clearest role when diuresis or an additional antihypertensive agent is also a desired effect</li><li>• In between-study comparisons, high-dose furosemide was more kaliuretic than metolazone in patients with GFR &lt;20 ml/min per 1.73 m<sup>2</sup><sup>214,216</sup></li></ul>

Mineralocorticoid agonists	<ul style="list-style-type: none"> <li>• Dependent on kidney function</li> <li>• Weak (small observational studies and clinical trials) and inconsistent data about efficacy<sup>217,218</sup></li> <li>• Possibly harmful, given the hypothesis that mineralocorticoid antagonism may reduce CV outcomes in ESKD</li> </ul>
Gastrointestinal potassium wasting	<ul style="list-style-type: none"> <li>• Potential management option</li> <li>• Scant evidence</li> <li>• One small pre-post study<sup>219</sup> found that increasing the number of stools from 1 to 2–4 per day with laxatives lowered potassium from mean <math>5.9 \pm 0.2</math> to <math>5.5 \pm 0.2</math> mmol/l without inducing diarrhea</li> </ul>
Correction of coincident acidosis	<ul style="list-style-type: none"> <li>• No evidence</li> </ul>
Use of low potassium dialysate	<ul style="list-style-type: none"> <li>• Observational evidence of increased risk of mortality, arrhythmias and emergency department visits at dialysate potassium concentration <math>&lt;2</math> mmol/l and with higher serum-dialysate gradients (see text)</li> </ul>
Older potassium binder: SPS	<ul style="list-style-type: none"> <li>• Concern about rare but serious adverse gastrointestinal effects from postmarketing studies</li> <li>• FDA warning in 2009 against use with sorbitol<sup>220</sup></li> <li>• Use only in patients with normal bowel function</li> <li>• Limited randomized evidence for efficacy</li> <li>• Binds other medications; other oral medications to be taken at least 3 h before or 3 h after SPS, 6 hours in patients with gastroparesis<sup>221</sup></li> </ul>
Newer potassium binders: patiromer, zirconium cyclosilicate	<ul style="list-style-type: none"> <li>• Evidence for efficacy in reducing hyperkalemia incidence of up to 12 mo</li> <li>• Evidence of adverse effects for exposure of up to 12 mo</li> <li>• Lack of large-scale postmarketing studies</li> <li>• Patiromer binds other medications; other oral medications to be taken at least 3 h before or 3 h after patiromer<sup>222</sup></li> <li>• Zirconium cyclosilicate affects the absorption of drugs whose bioavailability is dependent on gastric pH<sup>a</sup>; these oral medications should be taken at least 2 h before or 2 h after zirconium cyclosilicate<sup>223</sup></li> </ul>

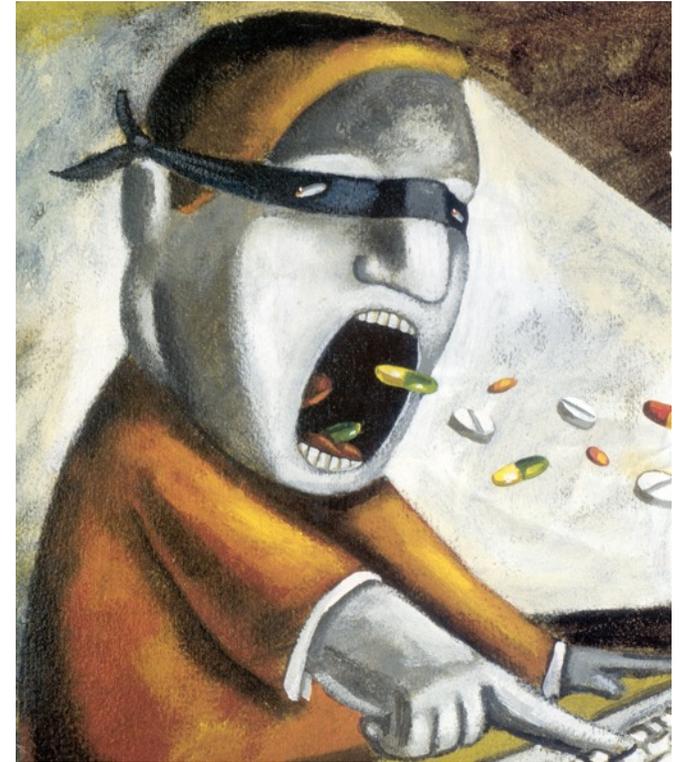
CV, cardiovascular; ESKD, end-stage kidney disease; FDA, US Food and Drug Administration; GFR, glomerular filtration rate; PD, peritoneal dialysis; RAAS, renin-angiotensin-aldosterone system; RCT, randomized controlled trial; SPS, sodium polystyrene sulfonate.

Conference participants were unable to provide evidence-based recommendations or suggestions on preferential strategies because of a lack of evidence for most of the strategies, the absence of evidence on comparative efficacy of alternative strategies, and the potential for harm with at least some of them.

<sup>a</sup>Zirconium cyclosilicate interferes with the absorption of drugs that exhibit pH-dependent bioavailability, e.g., atorvastatin; the azole antifungals ketoconazole, itraconazole, and posaconazole; dabigatran; furosemide; some drugs for HIV (atazanavir, nelfinavir, indinavir, ritonavir, saquinavir, raltegravir, ledipasvir, and rilpivirine); and the tyrosine kinase inhibitors (erlotinib, dasatinib, and nilotinib). We were unable to identify a comprehensive reference permitting clinicians to identify whether an individual drug has gastric pH-dependent bioavailability. In 2018 the FDA initiated a process to improve approaches to the assessment of this issue.<sup>224</sup>

# Reduce the ACE-I/ARB

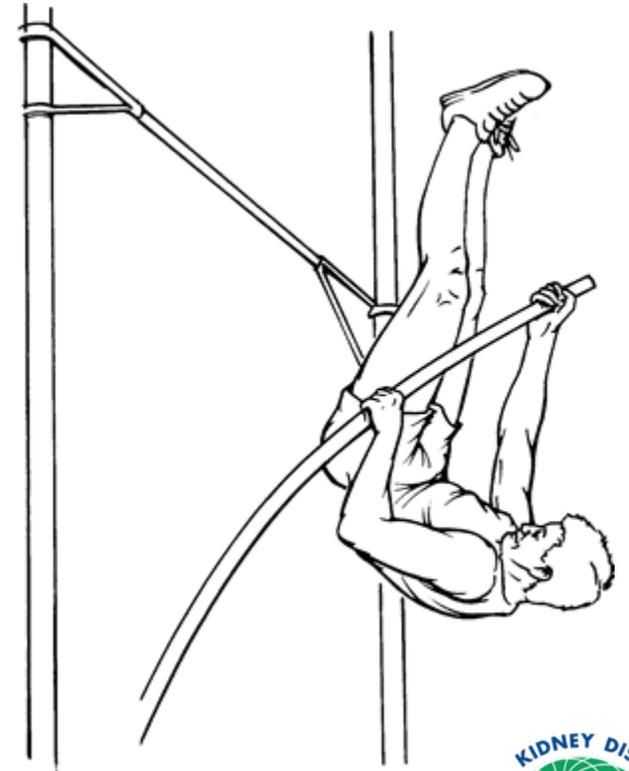
- Problematic
- Pre 2013: Combination ACE-I and ARBs
- Post 2014: ACE-I or ARB with spironolactone
- 2015: restore the combination therapy: And what about spironolactone?



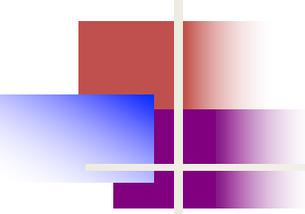
# When do you get concerned about potassium?

- Are you cardiology or nephrology?
- Acute or chronic?
- Background CKD

5.5 vs 6.0 mEq/L



# Are We Optimally Managing Patients With High Potassium?



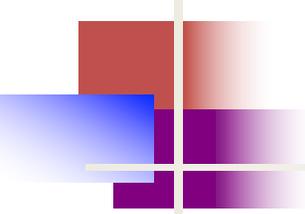
Change RAASi?

**KDIGO**

Treat high K?

Do nothing?





# Today's talk.....

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## ■ Treatment options

- What about the foods?

- Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia

- **What is on the horizon to lower potassium?**

- Unmet need of hyperkalemia in CKD: How would you manage the patient?

# Current Treatment Options for Hyperkalemia are Limited

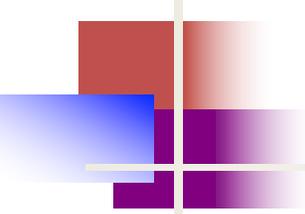
- ◆ Most acute therapies do not remove excess potassium, and impractical in outpatient setting
  - IV calcium, sodium bicarbonate, insulin and dextrose, nebulized beta-adrenergic agonists
- ◆ Dietary potassium restriction met by non-adherence, limits healthy food choices
- ◆ Sodium polystyrene sulfonate (SPS)
  - Uncertain efficacy - no rigorous clinical trials
  - Poorly tolerated
  - Reports of serious intestinal toxicity

**There is a clinical need for a hyperkalemia treatment that is effective, safe and well-tolerated**

# Key Characteristics of Old and New K<sup>+</sup>-Binding Agents

	Sodium Polystyrene Sulphonate	Patiromer	Sodium Zirconium Cyclosilicate
MOA <sup>1</sup>	Nonspecific cation binding in exchange for sodium	Patiromer is a polymer exchange resin	Selective K <sup>+</sup> binding in exchange for sodium and hydrogen
Time to Normokalemia	Unconfirmed	Within 1 week <sup>2</sup>	Within 24 hours for 84% of patients <sup>3</sup>
Onset of action	Unknown (generally hours to days)	7 hours after first dose <sup>4</sup>	1 hour following the first dose <sup>3</sup>
Drug–drug Interactions	With antacids, laxatives, digitalis, sorbitol, lithium, and thyroxine <sup>5</sup>	FDA: Must be taken 3 hours apart from other oral drugs <sup>6</sup>	Should be given 2 hours apart from oral medication with gastric pH-dependent bioavailability <sup>7</sup>
Location of K <sup>+</sup> Binding	Colon	Predominantly distal colon	Likely entire GI tract
Safety / Tolerability	Associated with: <ul style="list-style-type: none"> <li>•Safety and tolerability concerns<sup>8</sup></li> <li>•Electrolyte disturbances</li> </ul>	<ul style="list-style-type: none"> <li>•Hypomagnesaemia<sup>9</sup></li> <li>•GI side effects, e.g.</li> <li>•mild-to-moderate constipation</li> </ul>	<ul style="list-style-type: none"> <li>•Mild-to-moderate GI effects<sup>10</sup></li> <li>•Oedema</li> </ul>

FDA, US Food and Drug Administration; GI, gastrointestinal; MOA, mechanism of action; SPS, sodium polystyrene sulphonate; SZC, sodium zirconium cyclosilicate  
 1. Garimella PS, et al. Am J Kidney Dis 2016;67:545–547; 2. Weir MR, et al. N Engl J Med 2015;372:211–221; 3. Kosiborod M, et al. JAMA 2014;312:2223–2233; 4. Bushinsky DA, et al. Kidney Int 2015;88:1427–1433; Sanofi-Aventis. Kayexalate Prescribing information 2009; 6. Patiromer Prescribing Information 2016; 7. AstraZeneca. Sodium Zirconium Cyclosilicate Summary of Product Characteristics 2018;; 8. Lepage L, et al. Clin J Am Soc Nephrol 2015;10:2136–2142; 9. Bakris GL, et al. JAMA 2015;314:151–161; 10. Packham DK, et al. N Engl J Med 2015;372:222–231



# Today's talk.....

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## ■ Treatment options

- What about the foods?
- Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia
- What is on the horizon to lower potassium?

**■ Unmet need of hyperkalemia in CKD: How would you manage the patient?**

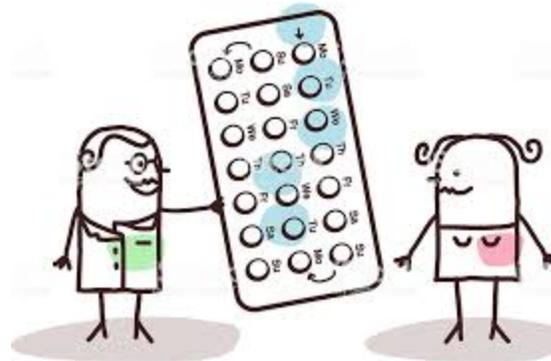


RAASi = Renin Angiotensin Aldosterone System inhibitors  
MRA = Mineralocorticoid Receptor Antagonists

# How would you manage the patient?

- Dietary restriction
- Reduce the dose of RAASi
- Correct the metabolic acidosis
- Increase diuretics
- Maybe regular SPS/or fludrocortisone

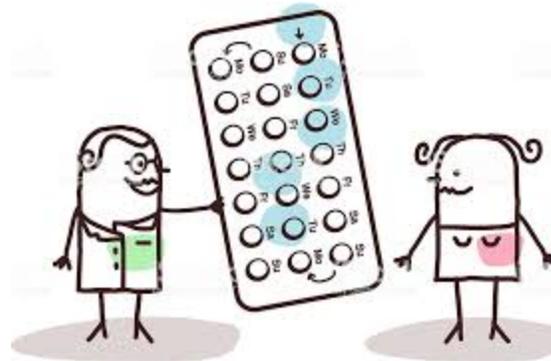
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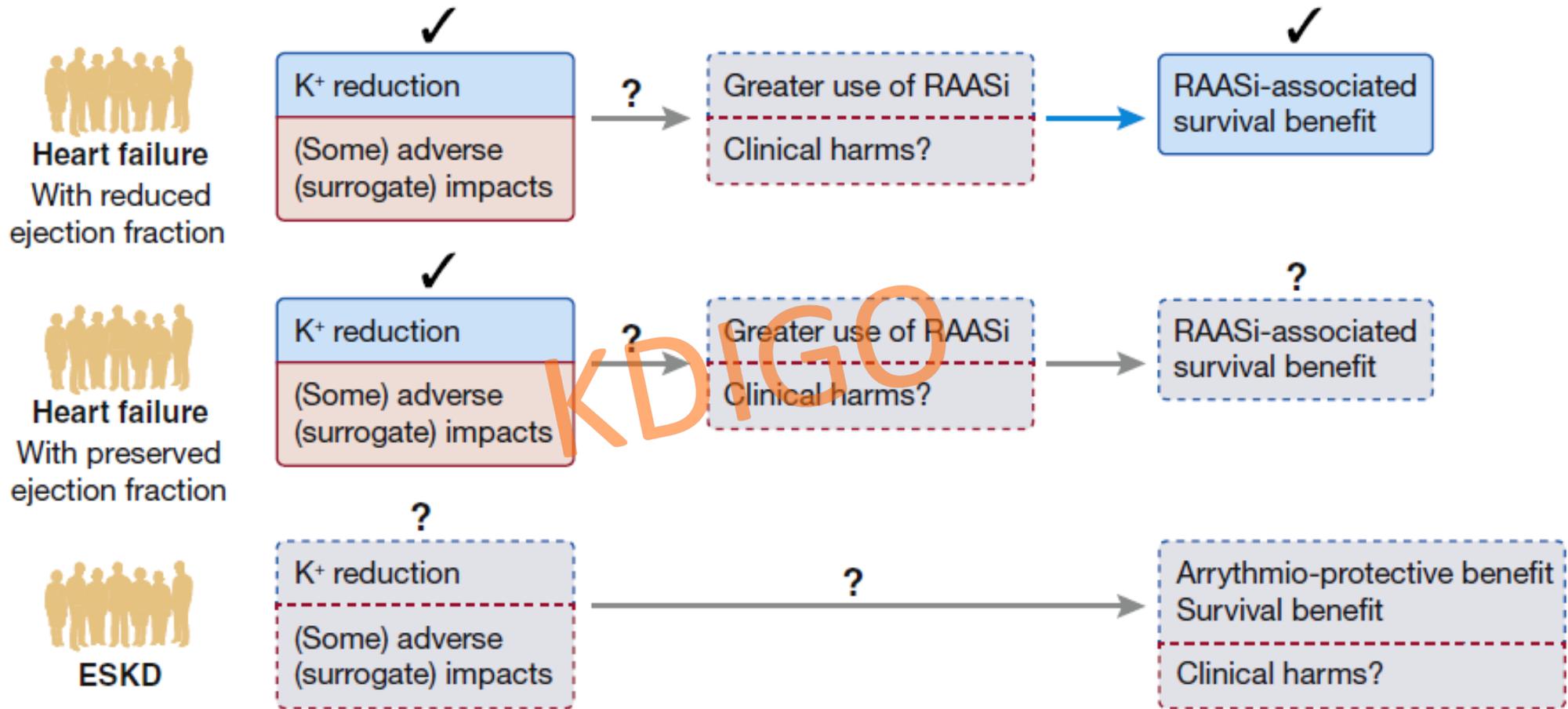


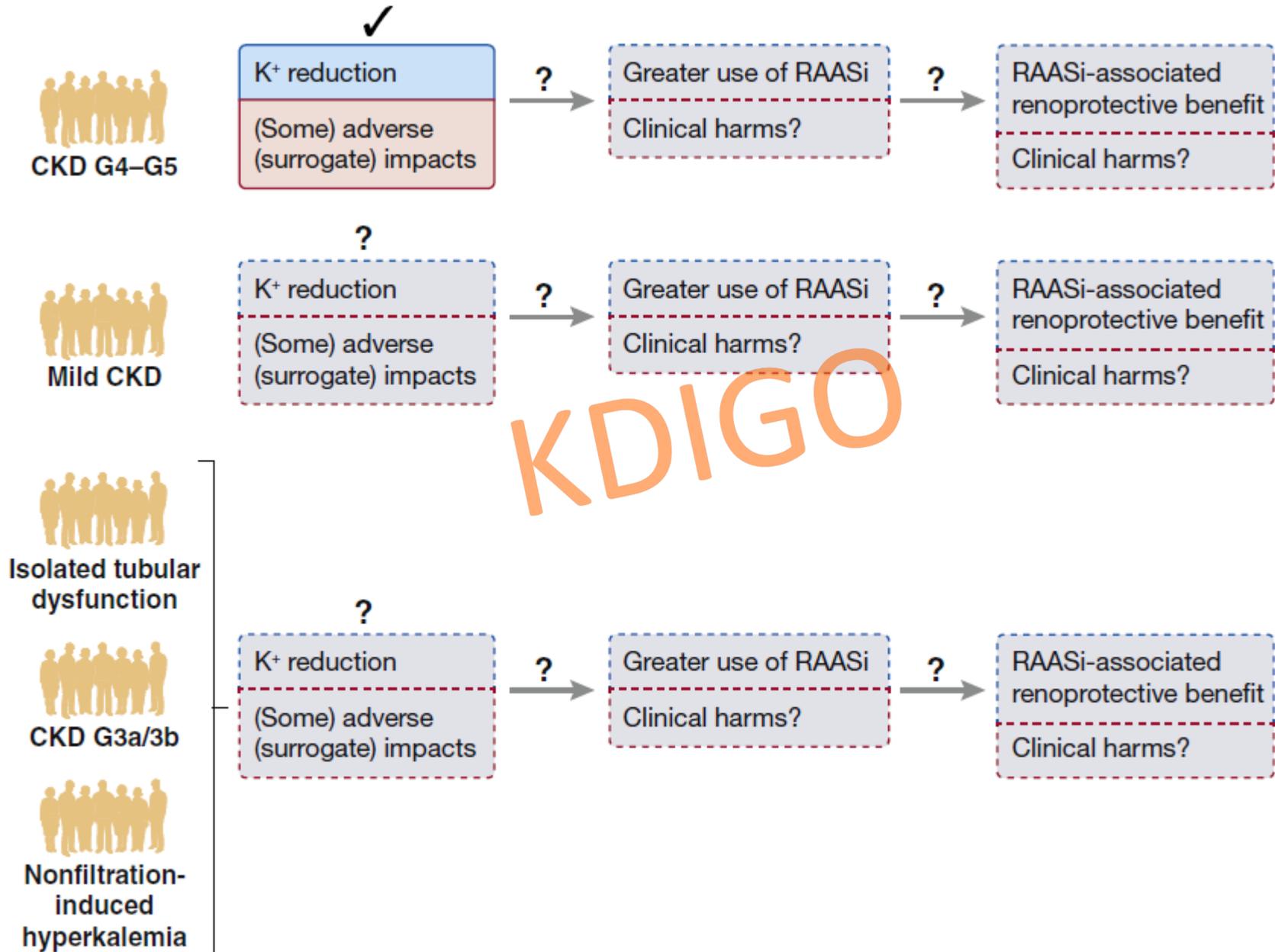
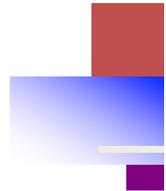
# How would you manage the patient?

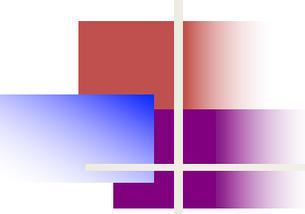
- Dietary restriction
- Reduce the dose of RAASi
- Correct the metabolic acidosis
- Increase diuretics
- Maybe regular SPS/or fludrocortisone
- Maintain the diet
- Maintain the maximum/optimal dose of RAASi
- Maintain the diuretics
- Start one of the newer potassium lowering binders

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# Today's talk.....

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- Treatment options
  - What about the foods?
  - Sub-optimal RAASi and MRA therapy due to fear of hyperkalemia
  - What is on the horizon to lower potassium?
- Unmet need of hyperkalemia in CKD: How would you manage the patient?

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RAASi = Renin Angiotensin Aldosterone System inhibitors  
MRA = Mineralocorticoid Receptor Antagonists