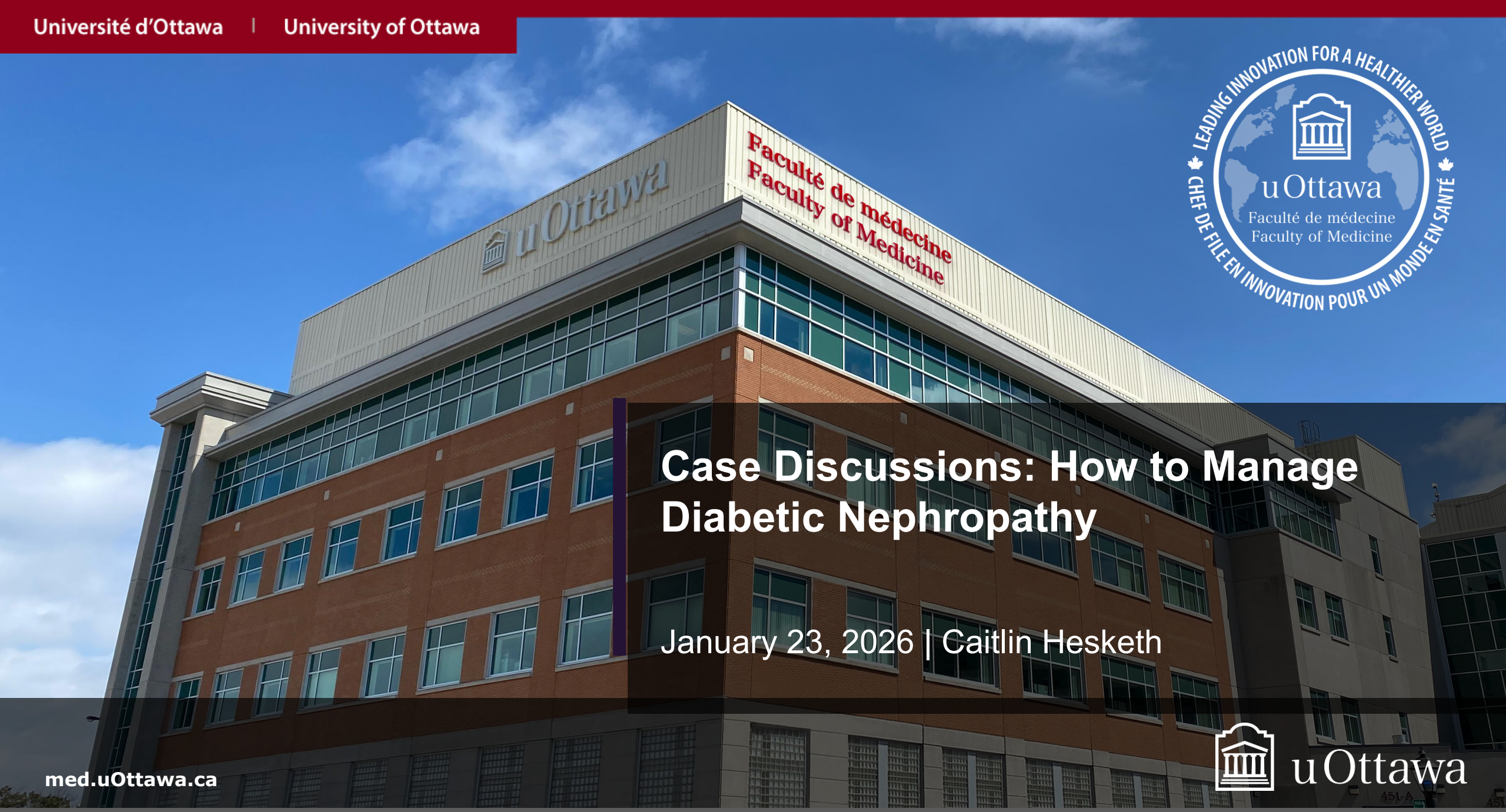


LEADING INNOVATION FOR A HEALTHIER WORLD
uOttawa
Faculté de médecine
Faculty of Medicine
CHEF DE FILE EN INNOVATION POUR UN MONDE EN SANTÉ

Case Discussions: How to Manage Diabetic Nephropathy

January 23, 2026 | Caitlin Hesketh

uOttawa

PRESENTER DISCLOSURE

- **Presenter:** Caitlin Hesketh

- **Relationships with commercial interests:**
  - **Grants/Research Support:** None
  - **Speakers Bureau/Honoraria:** Bayer, GSK, Otsuka
  - **Consulting Fees:** None
  - **Other:** n/a

LEARNING OBJECTIVES

1) **Review medication management after AKI**

2) **Assessment and management of hyperkalemia associated with CKD**

3) **Discuss use of MRAs in diabetic kidney disease**

4) **Summarize the four pillars of treatment in diabetic kidney disease**

Two years later

- Unfortunately, he had perforated appendix requiring OR and ICU stay; suffered AKI from ATN that did not fully resolve.
- Comes to you 1 month post discharge with med changes:
  - Candesartan 32 mg daily – stopped during admission (AKI)
  - Dapagliflozin 10 mg daily – stopped during admission (fear of euglycemic DKA)
  - Metformin 1000 mg twice daily – stopped during admission (AKI)
  - Continues on semaglutide 1 mg subcut every 1 week only

At your clinic:

- BP 150/83
- HbA1C 7.6%
- Creatinine 170 µmol/L – stable since discharge, eGFR 41 mL/min
- K 4.5
- ACR 110 mg/mmol

- What do you do with his candesartan and dapagliflozin?

Medication management post AKI

- RAAS inhibitor resumption after AKI recovery is associated with improved long term outcomes and little to no increased risk of recurrent AKI.
- Observational studies suggest increased mortality in patients who do not resume/start RAAS inhibitors post AKI.
- Recommendations are to restart RAAS inhibitor at lower doses (e.g. 8-16 mg) with monitoring for hyperkalemia and hypotension.
  - Resume dapagliflozin at separate visit, not same time.

Follow up 3 months post AKI:

- Now back on candesartan 16 mg and dapagliflozin
- BP 141/78
- HbA1C 7.2%
- Creatinine 192 µmol/L, eGFR 35 mL/min
- Na 140, **K 5.6**, HCO3 22, glucose 7
- ACR 80 mg/mmol

What do you do?

1. Do nothing and repeat electrolytes in 2 weeks
2. Send to emerg
3. Stop candesartan
4. Take away his bananas and potatoes
5. Add thiazide diuretic

Hyperkalemia - Treat, don't stop!

Address reversible contributors: constipation, metabolic acidosis, hyperglycemia

Ensure NSAID avoidance

Dietary interventions

Add diuretics, potassium exchange resins

Don't stop the RAAS inhibitor until you've tried these things!

Don't blame bananas

Potassium rich diets are full of healthy fruits and vegetables

Potassium supplementation, at a general population level, reduces BP and stroke risk

Low potassium intake is associated with lower survival in patients with normal AND reduced GFR

In people with CKD, estimations of dietary potassium correlate poorly with serum potassium

What is better?

Avoid processed foods

Avoid potassium additives

Excellent glycemic control

Get enough fiber

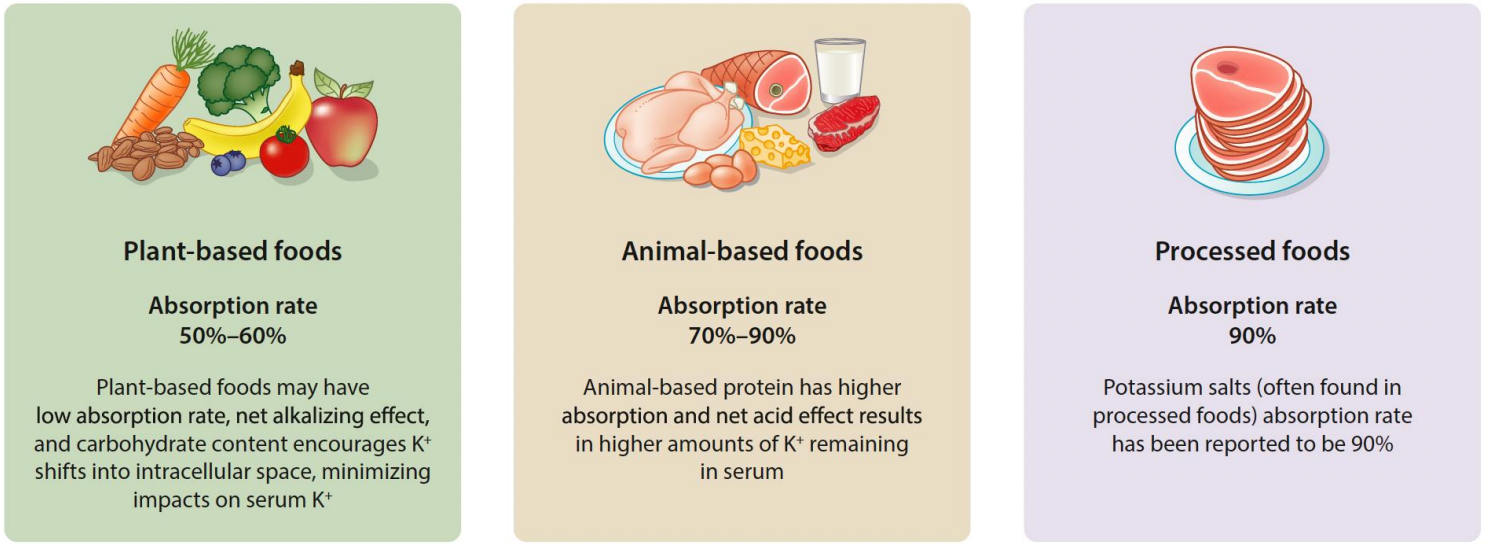


Figure 33 | Potassium absorption rates of plant-based, animal-based, and processed foods. Data from Picard K, Griffiths M, Mager DR, Richard C. Handouts for low-potassium diets disproportionately restrict fruits and vegetables. *J Ren Nutr*. 2021;31:210–214.[592]

Diuretics and potassium

- There is RCT evidence to support thiazide use in HTN and stage IV CKD (CLICK trial, Agarwal *et al*. N Engl J Med 2021;385:2507-2519).
- Indapamide 2.5 mg may lower serum K by ~ 0.3 to 0.4 mmol/L
- Added benefit of reducing proteinuria, improving BP control, reduced LV mass (indapamide).

Rousch et al. Hypertension 2020; 65(5):1041-1046

Next steps

- You add indapamide 2.5 mg once daily
- Seeing him again 4 weeks later:
- BP 128/72
- HbA1C 7.3%
- Creatinine 200 µmol/L, eGFR 34 mL/min
- Na 136, K 4.8, HCO3 24, glucose 7.5
- ACR 55 mg/mmol

Current meds:
Candesartan 32 mg once daily
Indapamide 2.5 mg once daily
Semaglutide 1 mg once weekly
Dapagliflozin 10 mg once daily

Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012

GFR categories (ml/min/1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60–89			
	G3a	Mildly to moderately decreased	45–59			
	G3b	Moderately to severely decreased	30–44			*
	G4	Severely decreased	15–29			
	G5	Kidney failure	<15			

Patient risk of progression to kidney failure requiring dialysis or transplant:

AT 2 YEARS	AT 5 YEARS
9.5 %	26.78 %

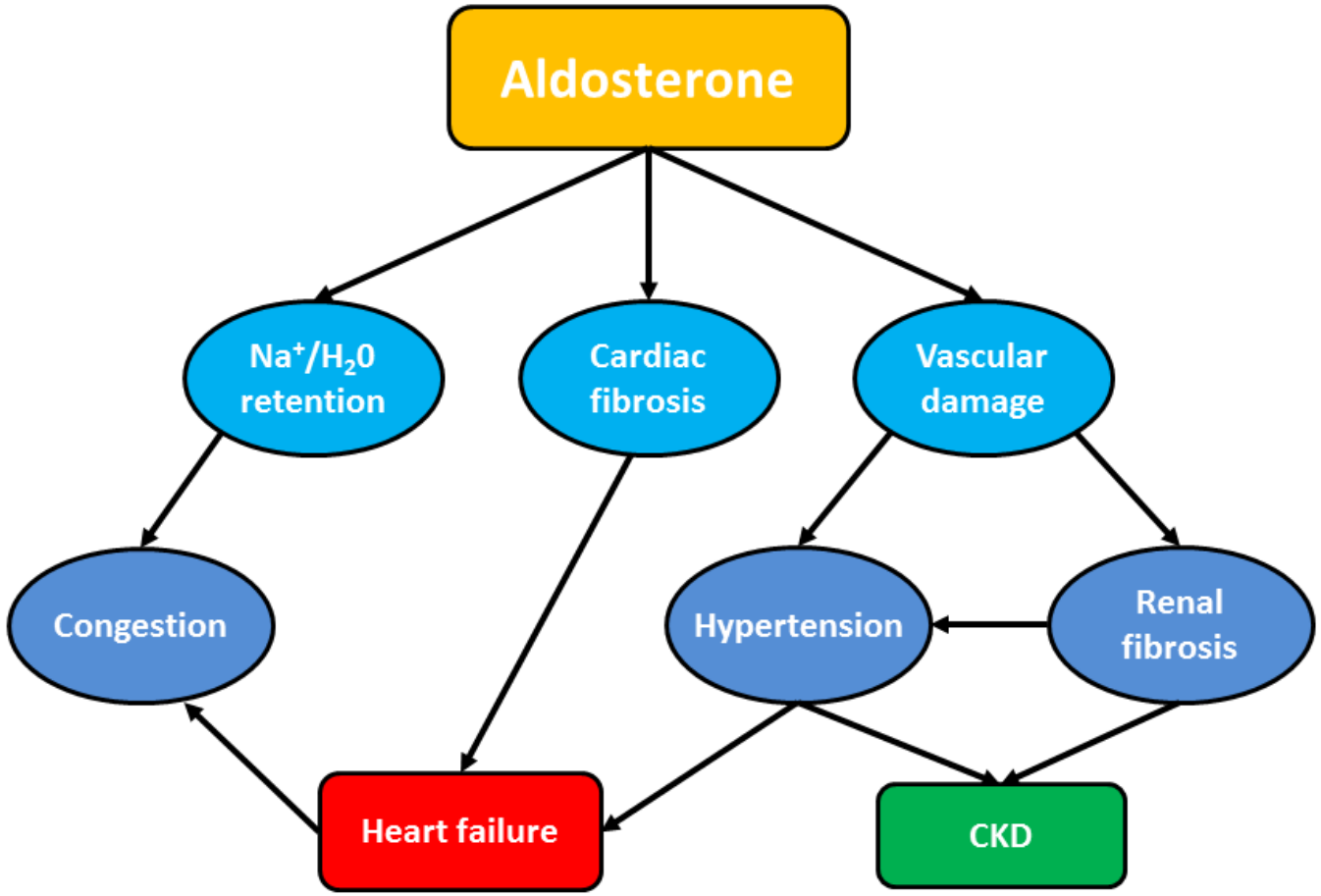
Risk thresholds used in health systems include:

- 3-5 % over 5 years for referral to a kidney doctor
- 10 % over 2 years for team based care (Kidney Doctor, Nurse, Dietician, Pharmacist)
- 20-40 % over 2 years for planning a transplant or fistula

What should we do next to further slow this man's progression to ESRD?

1. Counsel him on starting low protein diet (<0.6 g/kg/d)
2. Add finerenone 10 mg once daily
3. Stop indapamide
4. Sick day medication counselling
5. Nothing – he is optimized!

MR antagonists



Prior data for Spironolactone/Eplerenone

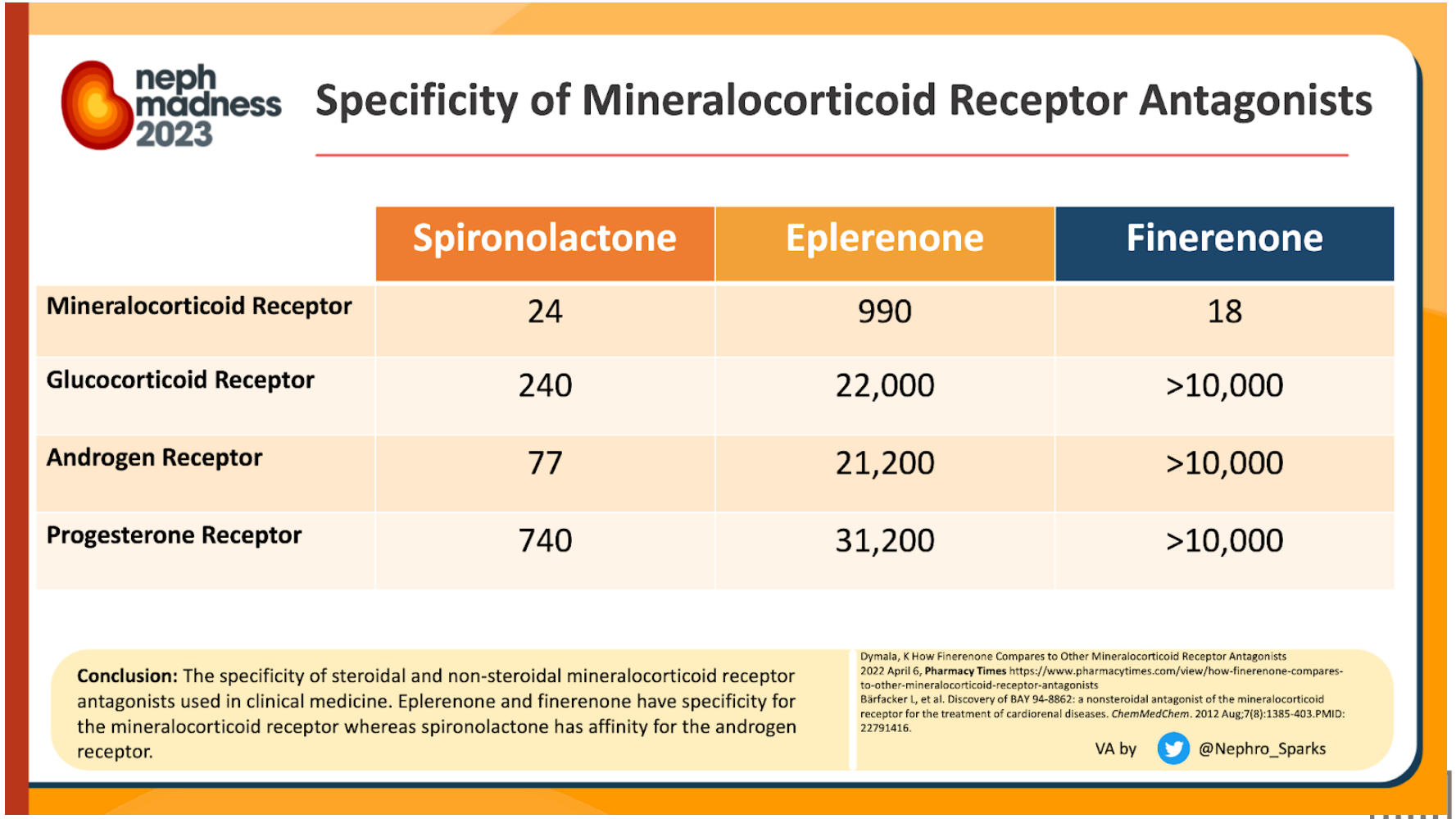
Small studies
No hard kidney end-points

Pro: Reduce proteinuria

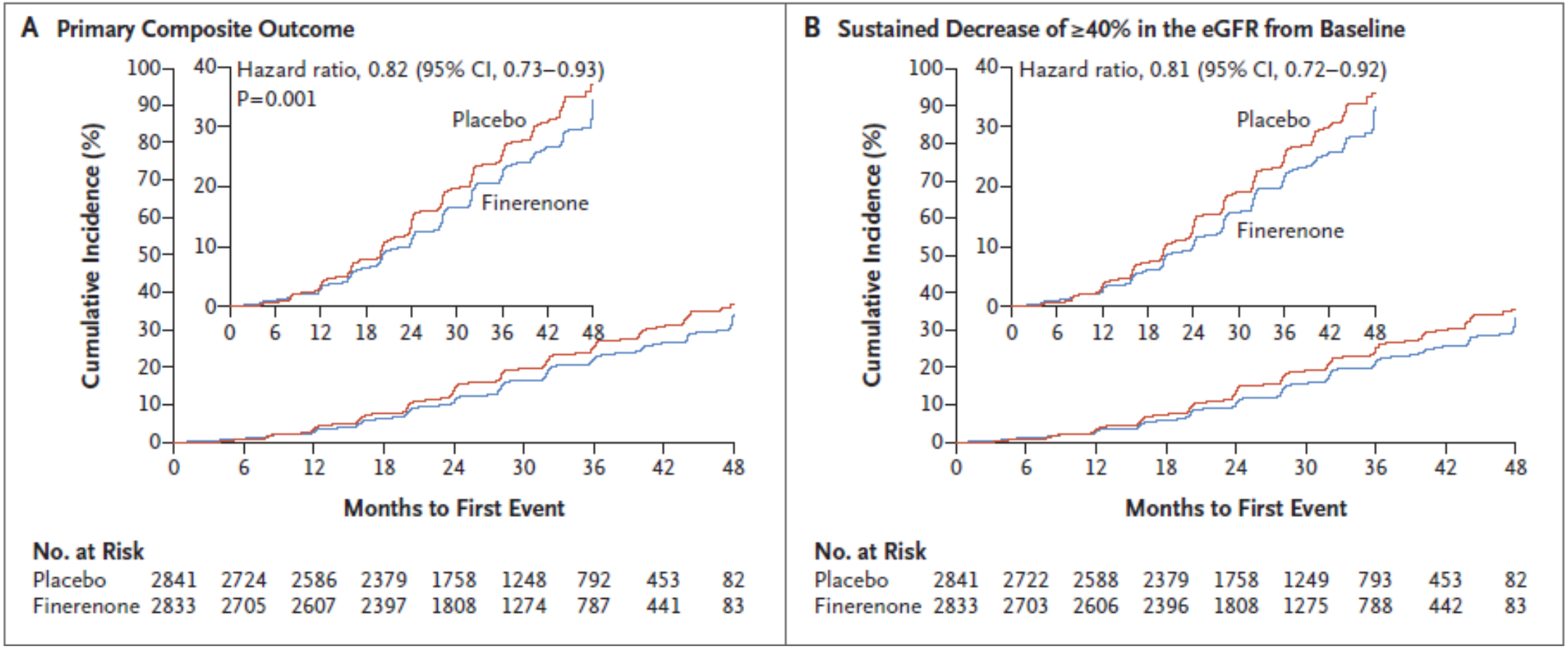
Cons: Hyperkalemia (RR 2.0), gynaecomastia (RR 5.1)

Bolignano et al. Cochrane Database Syst Rev. 2014 Apr 29:(4):CD007004.

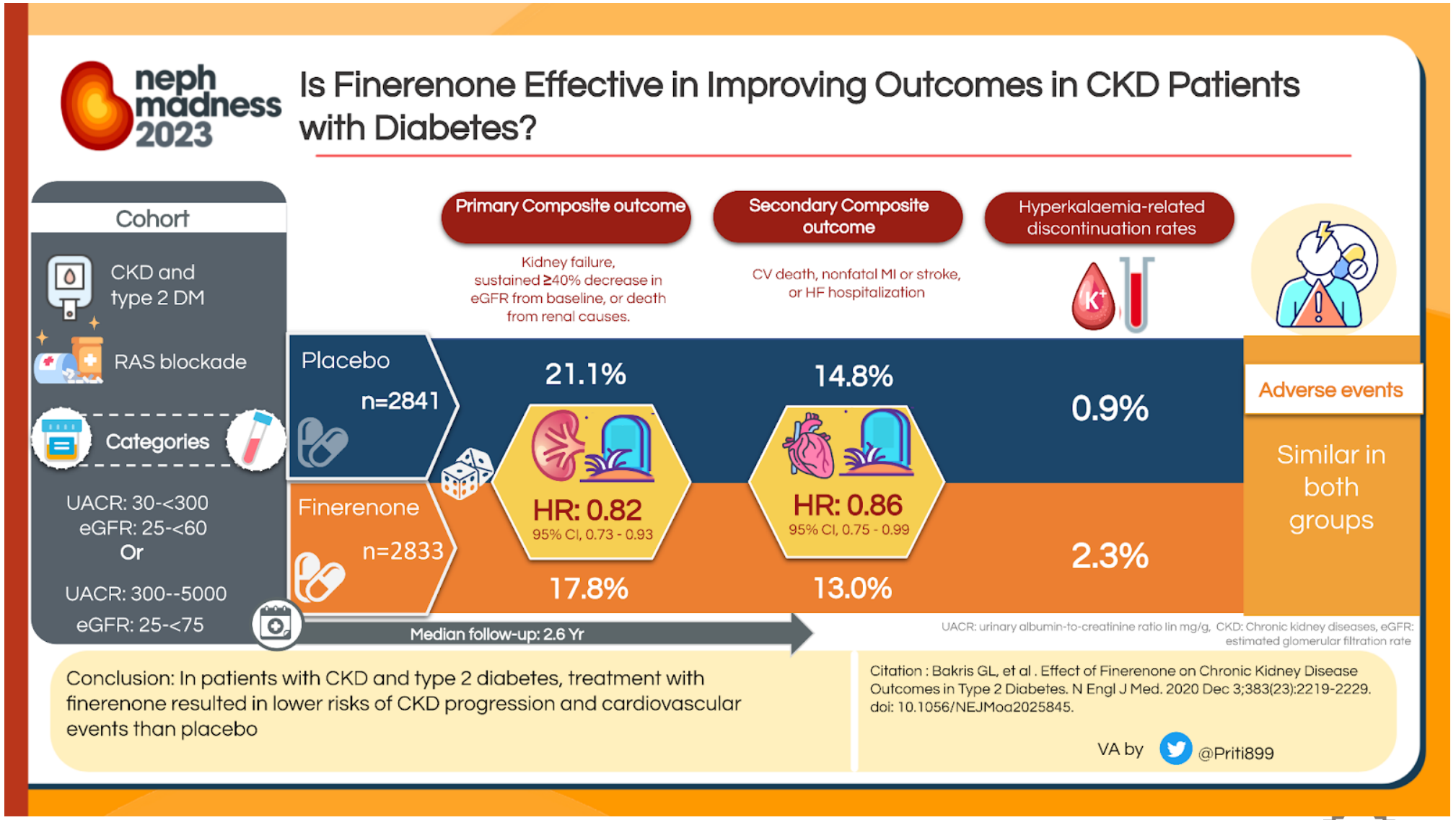
Finerenone: More than just "fancy spironolactone"



FIDELIO Trial



Bakris *et al*. NEJM 2020; 383(23):2219-2229

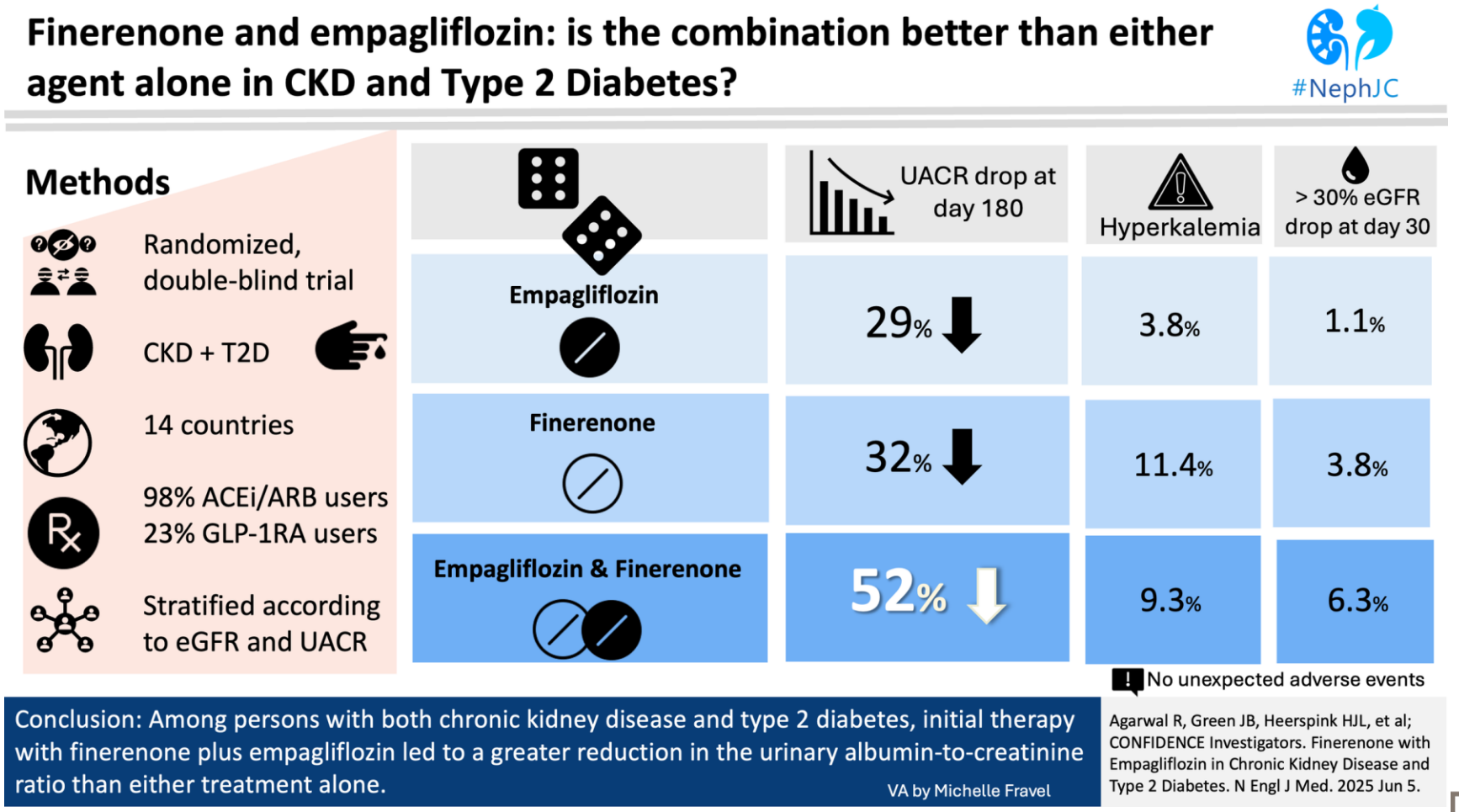

neph madness 2023
Is Finerenone Effective in Improving Outcomes in CKD Patients with Diabetes?
Cohort
CKD and type 2 DM
RAS blockade
Categories
UACR: 30-<300
eGFR: 25-<60
Or
UACR: 300--5000
eGFR: 25-<75
Primary Composite outcome
Kidney failure, sustained ≥40% decrease in eGFR from baseline, or death from renal causes.
Secondary Composite outcome
CV death, nonfatal MI or stroke, or HF hospitalization
Hyperkalaemia-related discontinuation rates
Adverse events
Similar in both groups
Placebo
n=2841
21.1%
14.8%
0.9%
Finerenone
n=2833
HR: 0.82
95% CI, 0.73 - 0.93
HR: 0.86
95% CI, 0.75 - 0.99
17.8%
13.0%
2.3%
Median follow-up: 2.6 Yr
UACR: urinary albumin-to-creatinine ratio in mg/g, CKD: Chronic kidney diseases, eGFR: estimated glomerular filtration rate
Conclusion: In patients with CKD and type 2 diabetes, treatment with finerenone resulted in lower risks of CKD progression and cardiovascular events than placebo
Citation : Bakris GL, et al . Effect of Finerenone on Chronic Kidney Disease Outcomes in Type 2 Diabetes. N Engl J Med. 2020 Dec 3;383(23):2219-2229. doi: 10.1056/NEJMoa2025845.
VA by @Priti899

Prescribing Finerenone

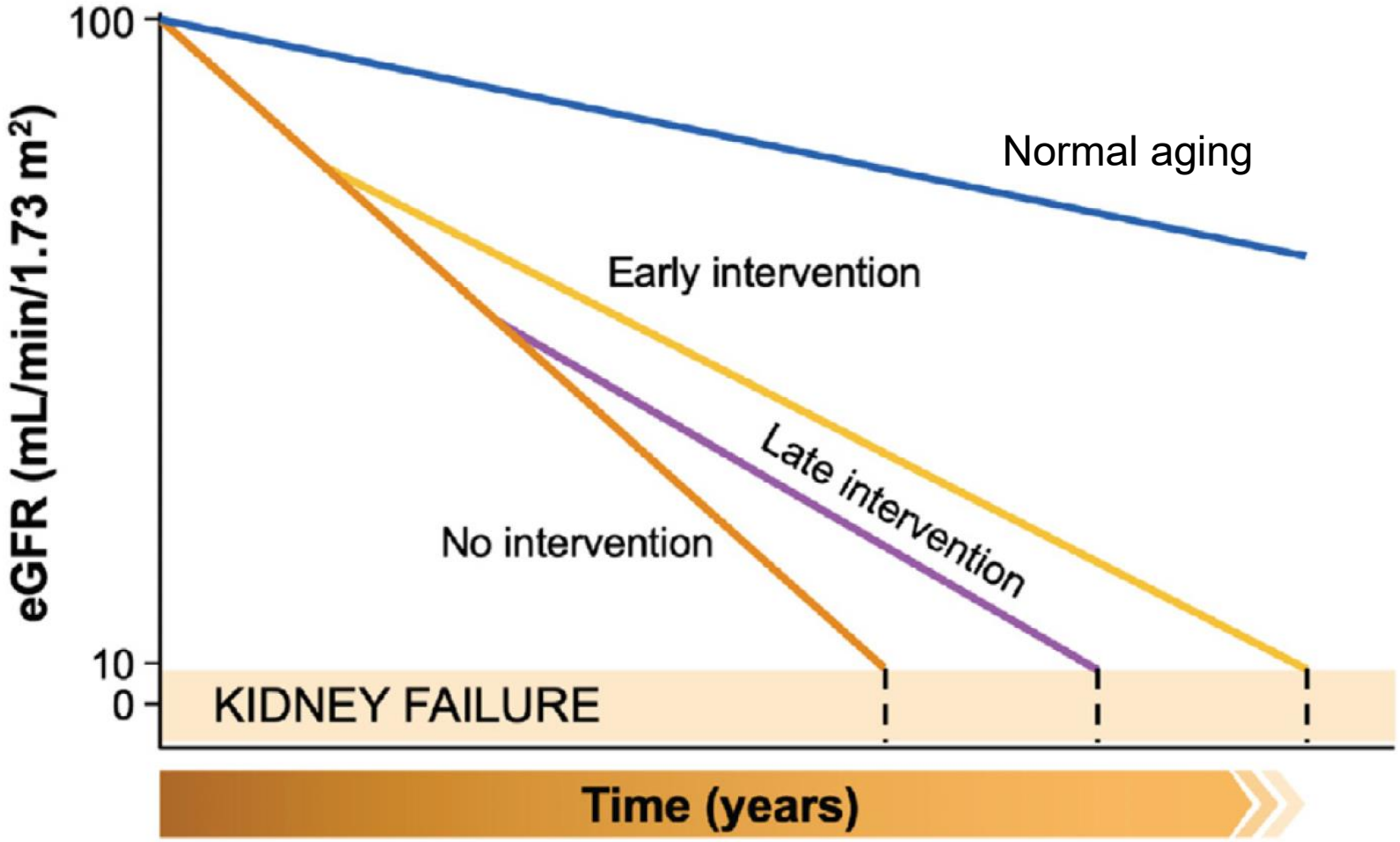
- Indications: patients with T2DM + Albuminuria (ACR >3)
- Already on RAAS inhibitor at max tolerated dose
  - SGLT-2 may be helpful to reduce hyperkalemia
- ODB coverage (LU code 700)
  - otherwise ~ $3.25 a pill ~ $100/month
  - Most private insurance will cover it

7. Finerenone is prescribed in consultation with a nephrologist or other clinician with experience in the diagnosis and management of patients with CKD and T2D.

What if they're already on SGLT-2 inh?



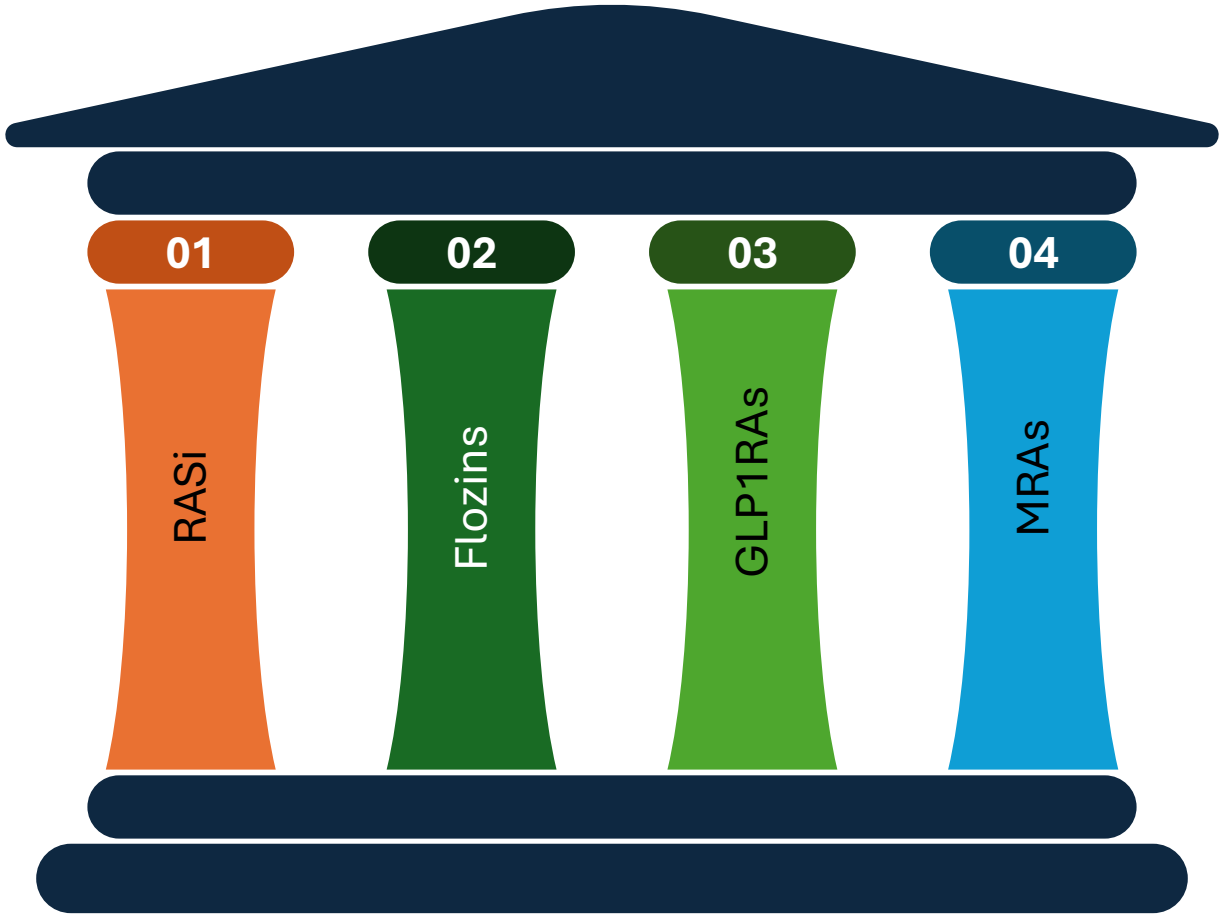
There is hope!



Tobe *et al*. Can J Diabetes 2025; 49:73-86

4 pillars of therapy

Do we have to do one after the other in a particular order?



Can we start all 4 agents at once?

Consider Flozins early (either step 1 or 2)

Otherwise dictated by BP or BG

DM/Obesity/highish K: GLP1RAs first

RASi + Flozin + MRA all have GFR effect , so not a good idea!

Consider accelerated addition (time is nephrons)

MRA + Flozin may be reasonable

Stolen without permission from Swapnil Hiremath

Summary

- Diabetic kidney disease historically has poor renal outcomes; however, there is renewed optimism that we can prevent kidney failure using the four pillars of treatment.
- Medications only work if they are prescribed and patients take them.
- Our next challenge will be how combine therapies and implementation.
- Treating diabetic kidney disease is a team sport!