CME OUTFITTERS

Improving Early Identification and Integrated Management

of Cardio-Renal-Metabolic Risk in Family Medicine



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To submit a question, please go to the *Ask Question* tab at the bottom of the screen.



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Consultant—Abbott; Bayer; Novo Nordisk; and Sanofi

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All identified conflicts of interest have been mitigated.

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LEARNING OBJECTIVES

1. Identify the shared pathophysiologic mechanisms linking cardiovascular, kidney, and metabolic disease, including how these mechanisms inform integrated cardiorenal risk management in family medicine settings

2. Assess risk for CKD and cardiovascular complications using guideline-recommended screening tools (eGFR and uACR) to inform earlier intervention and referral decisions

3. Apply patient-centered, guideline-aligned management strategies that support multidisciplinary care, shared decision-making, and longitudinal monitoring across the CKM continuum

Why CKM, Why Family Medicine, Why Tonight

Jay H. Shubrook, DO, FAAFP, FACOFP



Audience Response



In your current practice, which aspect of cardiovascular-kidney-metabolic (CKM) risk is most difficult to recognize early in patients with type 2 diabetes?

A. Declining kidney function before it is symptomatic

B. Albuminuria – ordering it, or knowing what to do with the result

C. Early heart failure, especially with preserved ejection fraction

D. Nothing is hard to recognize; the hard part is finding time to act on it

CME OUTFITTERS

ARS POLLING RESULTS

Placeholder



Almost Every Adult in Your Panel Is Somewhere on This Continuum

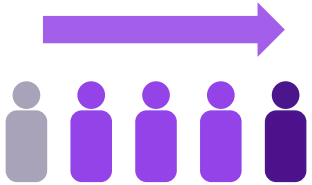
CKM stage 1-4 includes the vast majority of U.S. adults

90%

of U.S. adults are in CKM stage 1-4

Stage 0 (no CKM risk factors) is now the exception, not the baseline.

Stage 0 often progresses within 10 years



Stage 0 — Higher CKM stage within 10 years

Progression tracks with higher CVD risk and higher mortality.

Much of CKM remains unrecognized despite being common

9 in 10

Adults with CKD do not know they have it

Most CKD in the U.S. is attributable to T2D and hypertension, conditions family medicine already manages.

Overweight and obesity are common in U.S. adults

> 70%

of U.S. adults are overweight or have obesity

Overweight and obesity affect more than 70% of U.S. adults.

CKM is widespread, progressive, and often unrecognized, making primary care a critical intervention point.

CKD = chronic kidney disease; CVD = cardiovascular disease; T2D = type 2 diabetes.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Centers for Disease Control and Prevention [CDC]. https://nccd.cdc.gov/ckd.

What the 2026 CKM Guideline Adds

From Concept to Numbers

The 2023 AHA advisory named CKM syndrome. The 2026 guideline puts numbers on it.

2023 AHA advisory → 2026 CKM guideline

A True Multisociety Guideline

First multisociety guideline focused on CKM

AHA · ACC · ADA · ASN

Retires and replaces the 2013 overweight and obesity guideline.

Actionable in the Family Medicine Visit

Three key updates you can use in practice:

Staging
Numeric criteria you can apply at the desk.

PREVENT
Risk number informs which therapies to prioritize.

eGFR + uACR
Recommended annually from CKM stage 2 onward (COR 1).

What It Does Not Do

The guideline does not issue its own blood pressure or lipid recommendations; those defer to:

- **2025 blood pressure guideline**
- **2026 dyslipidemia guideline**

This guideline adds a unified, number-driven framework to identify risk, guide therapy, and integrate cardiovascular, kidney, and metabolic care into primary care.

AHA = American Heart Association; eGFR = estimated glomerular filtration rate; uACR = urine albumin-to-creatinine ratio.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Understanding the Shared Biology of CKM Syndrome

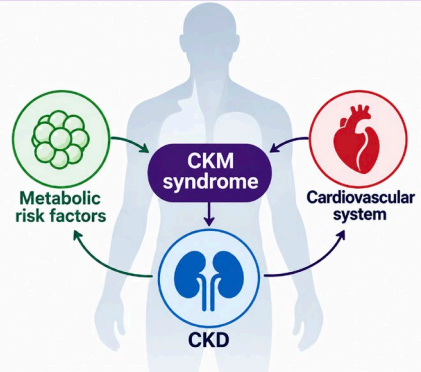
Chiadi E. Ndumele, MD, PhD



One Syndrome, Not Three Diagnoses That Happen to Coexist

The Guideline Definition

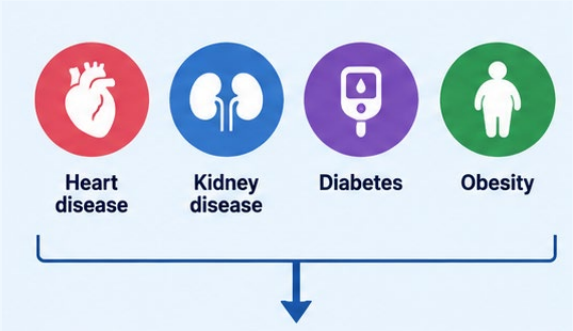
CKM syndrome is a **systemic disorder** characterized by pathophysiological interactions among metabolic risk factors, CKD, and the CV system, **leading to multiorgan dysfunction and a high rate of adverse CV outcomes.**



Connected systems. Greater risk. Multiorgan impact.

The Plain-Language Version for Patients

CKM syndrome is a health disorder due to **connections among heart disease, kidney disease, diabetes, and obesity,** leading to poor health outcomes.



These interconnected conditions can lead to premature mortality and other poor health outcomes.

Why the Framing Matters in Primary Care

Often managed separately

The conditions are managed in separate visits, by separate algorithms, against separate targets – while the underlying process is continuous.

A different approach

Treating them as one process changes what you look for, when you look, and what you reach for first.

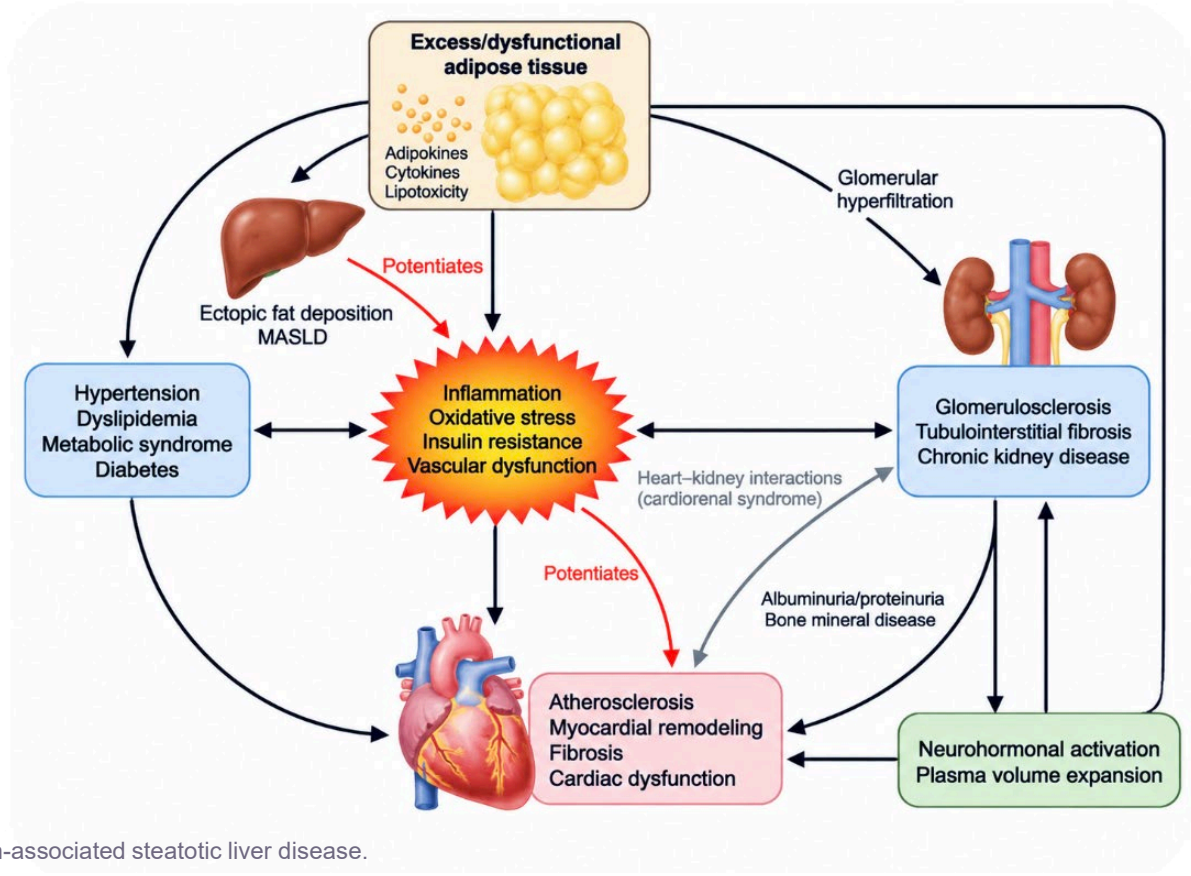
Broader factors play a role

CKM syndrome and its outcomes are shaped by policy, economic, and environmental conditions for self-care.

CV = cardiovascular.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

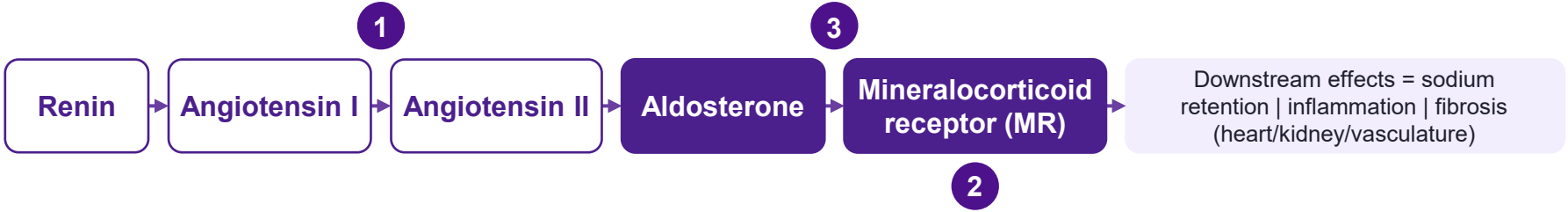
Adiposity as the Upstream Driver



MASLD = metabolic dysfunction-associated steatotic liver disease.

Adapted from Ndumele CE, et al. *Circulation*. 2023;148(20):1606-1635 and 1636-1664. Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

The Aldosterone Pathway: Three Places to Intervene



1 **WHERE IT ACTS**
Blocks angiotensin II generation or its receptor

CLASS
ACE inhibitors/ARBs

WHERE IT FITS TODAY
In every current CKM, CKD, and HF guideline.

2 **WHERE IT ACTS**
Blocks the mineralocorticoid receptor

CLASS
Nonsteroidal MRAs

WHERE IT FITS TODAY
Guideline-recommended in CKD + T2D and in HF with LVEF ≥ 40%.

3 **WHERE IT ACTS**
Blocks aldosterone synthesis upstream of the receptor

CLASS
Aldosterone synthase inhibitors

WHERE IT FITS TODAY
Emerging class. One agent FDA-approved for resistant hypertension. ***Not in CKM, CKD, or HF guidelines***. Phase III cardiorenal trials ongoing: **baxdrostat + dapagliflozin:** BaxDuo-CKD, BaxDuo-Pacific, Prevent-HF; **vicadrostat + empagliflozin:** EASI-HF Reduced, EASI-HF Preserved, EASi-PROTKT

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blockers; HF = heart failure; LVEF = left ventricular ejection fraction; MRA = mineralocorticoid receptor antagonist.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Brown NJ. *Hypertension*. 2008;51(2):161-167. Tuttle KR, et al. *Lancet*. 2024;403(10424):379-390.

How These Drivers Show Up in the Population

1 The risk factor mix has shifted, and old tools were built for a different population.

Improving

- Far wider use of antihypertensive and lipid-lowering therapy
- Declining tobacco use

Rising or persistent

- Rising obesity and CKD
- Persistently high burden of diabetes
- Rising triglyceride levels

2 Consequences for how we estimate risk:

- Pooled cohort equations **overestimated risk** when compared to observed event rates.
- **PREVENT** adds kidney and metabolic input to the model, and with development in contemporary cohorts demonstrates improved accuracy.

3 Consequences for who is in front of you:

- Heart failure (particularly HFpEF) is a **growing share of cardiovascular disease,** and it is the phenotype most likely to present first in primary care.
- The overlap between diabetes, kidney disease, and cardiovascular disease is now the **common case** rather than the complicated one.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889–e2007. Ndumele CE, et al. *Circulation*. 2023;148(20):1606-1635 and 1636-1664.

The Overlap Is the Rule, Not the Exception

Diabetes, CKD, and heart failure overlap heavily, and risk concentrates at the overlap.

How often they travel together:

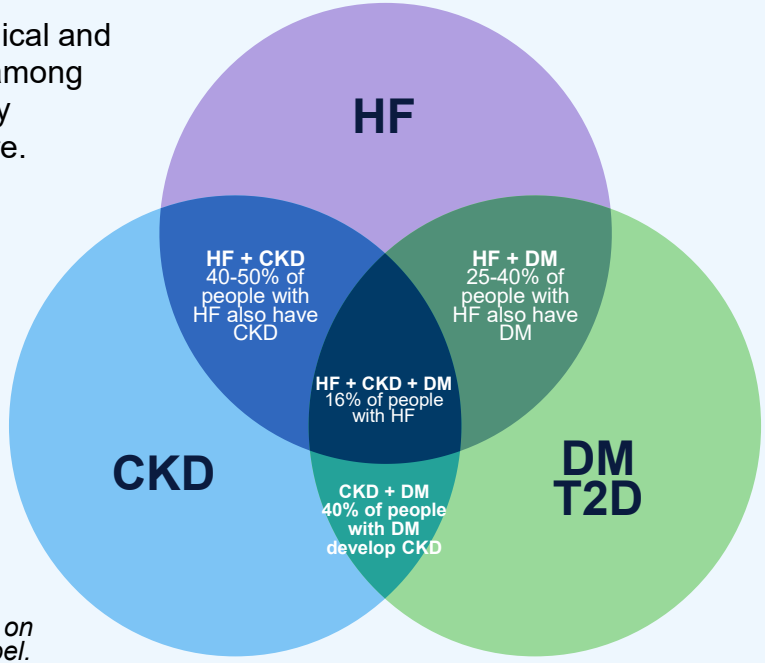
- 40% to 50% of people with HF also have CKD
- 25% to 40% of people with HF also have T2D
- 40% of people with T2D develop CKD
- 16% of people with HF have both CKD and T2D

The practical read:

The patient with two or three of these conditions is not simply a harder version of the same patient. They are a different risk category.

A Risky Intersection: Diabetes, Chronic Kidney Disease and Heart Failure

There is substantial clinical and epidemiologic overlap among diabetes, chronic kidney disease and heart failure.



Percentages are conditional on the group named in each label.

DM = diabetes mellitus.

Adapted from Vijay K, et al. *Cardiorenal Med.* 2022;12(1):1-10.

Outcomes Worsen as Conditions Accumulate

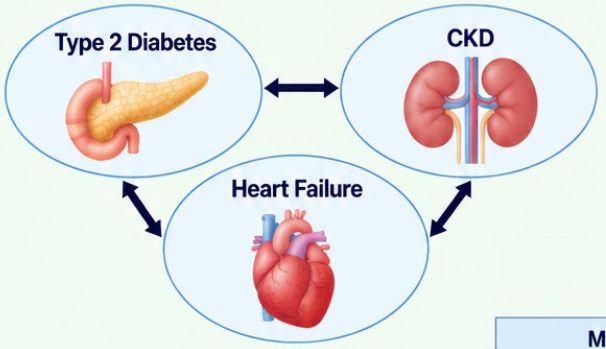
Each condition carries its own associations. What changes together is the magnitude.

- Stepwise increase in mortality as conditions accumulate
- Stepwise increase in cardiovascular and non-cardiovascular hospitalization

Among HF patients:

Having diabetes plus CKD carries the greatest risk of CV hospitalization, non-CV hospitalization, and all-cause mortality.

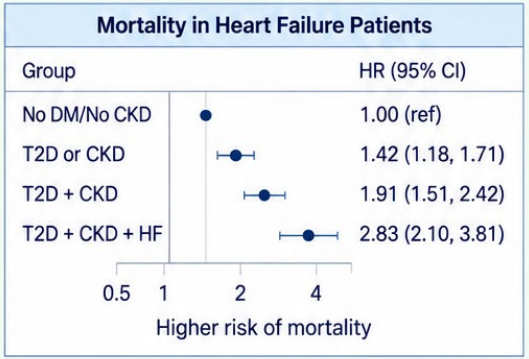
T2D, CKD, HF, and Outcomes



Among HF patients, the combination of DM + CKD is associated with the greatest risk for:

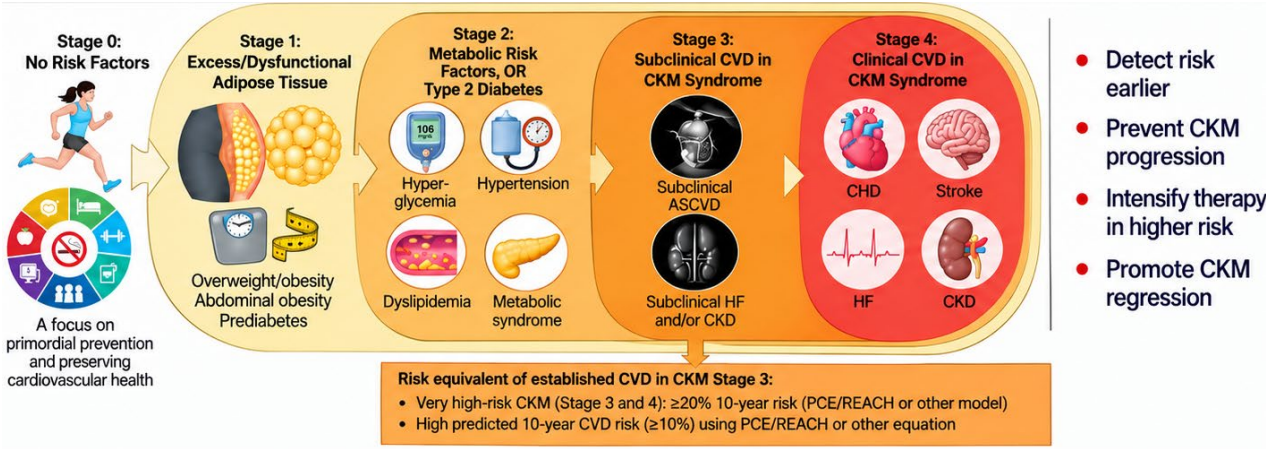
- CVD hospitalization
- Non-CVD hospitalization
- All-cause mortality

- Multidirectional associations among T2D, CKD and HF worsen clinical outcomes in HF:
  - **Type 2 DM:** 1.5 to 2-fold higher risk for CV death/HF hospitalization
  - **CKD:** 15% higher mortality for every 10 mL/min lower eGFR



CKM Staging: The Pathophysiology, Made Countable

0 Stage 0	1 Stage 1	2 Stage 2	3 Stage 3	4 Stage 4
No CKM risk factors	**Excess or dysfunctional adiposity**	**Metabolic risk factors and/or CKD**	**Subclinical CVD or risk equivalent**	**Clinical CVD**
Normal adiposity, glycemia, BP, lipids; no CKD or CVD	Overweight/obesity, abdominal adiposity, or prediabetes	Hypertension, hypertriglyceridemia, metabolic syndrome, T2D, or moderate- to high-risk CKD	Subclinical ASCVD/HF, very high-risk CKD, or PREVENT-CVD ≥ 20%	Established CVD; kidney failure may further define risk



ASCVD = atherosclerotic cardiovascular disease; BP = blood pressure; CHD = coronary heart disease; PCE/REACH = Pooled Cohort Equations/REduction of Atherothrombosis for Continued Health.

Adapted from Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Where the Windows of Opportunity Are

Staging is useful because it tells you what is still preventable

0-1 Stage 0 to 1

Before metabolic risk factors appear



- Adiposity is the target.
- Lifestyle modification first-line, aiming at 5% to 10% weight loss (COR 1, LOE A).

2 Stage 2

Risk factors and CKD present, no CVD yet



- The widest window in family medicine.
- Screening, risk quantification, and kidney-protective therapy all live here.
- Regression to an earlier stage is possible.

3 Stage 3

Subclinical disease



Still preventive, but the intervention is now about stopping progression to clinical events.

4 Stage 4

Clinical cardiovascular disease



Management, not prevention. PREVENT is no longer indicated at this stage.

Movement between stages goes both directions.

That is the argument for acting at stage 1 and 2.

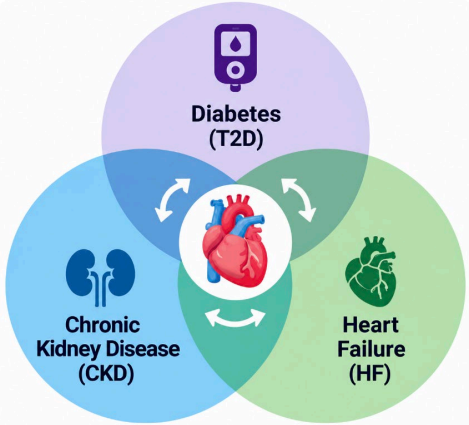
COR 1 = Class of Recommendation 1 (strong); LOE A = Level of Evidence A.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Heart Failure Inside the CKM Frame

What is growing

- **HFpEF and HFmrEF are the burgeoning phenotypes**
  - And among the most likely to present first in primary care
- **Symptoms are easy to attribute elsewhere**
  - Exertional dyspnea, fatigue, weight gain, or edema may be read as deconditioning, obesity, or age.
- **Pre-HF (subclinical HF) is CKM stage 3**
  - In patients without HF symptoms/signs: BNP ≥ 35 pg/mL or NT-proBNP ≥ 125 pg/mL, or structural/functional abnormalities on cardiac imaging.



A shared pathophysiology, with multidirectional risk and compounding outcomes.

The three-way interplay

- **Diabetes, CKD, and HF interact in more than one direction**
  - T2D raises HF risk even without prior myocardial infarction.
  - CKD independently raises HF hospitalization and cardiovascular mortality.
  - HF, in turn, accelerates kidney decline.
- **Clinical takeaway**
  - An abnormal uACR, glycemic markers, and HF risk belong in the same conversation.

U.S. guidance retains HFmrEF (LVEF 41% to 49%); the 2026 ESC heart failure guideline folds this group into HFrEF (LVEF < 50%).
BNP = B-type natriuretic peptide; ESC = European Society of Cardiology; HFmrEF = heart failure with mildly reduced ejection fraction; NT-proBNP = N-terminal pro–B-type natriuretic peptide.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Ndumele CE, et al. *Circulation*. 2023;148(20):1606-1635 and 1636-1664. Køber L, et al. *Eur Heart J*. 2026;ehag100.

PREVENT: Putting a Number on CKM Risk

Who to calculate

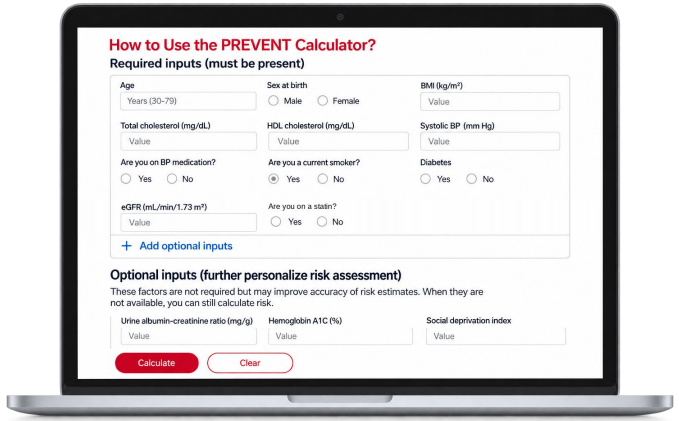
- In adults 30-79 without cardiovascular disease, calculate 10-year risk of total CVD, ASCVD, and HF using PREVENT (COR 1 | LOE B-NR)
- In adults 30-59, 30-year risk can also be useful (COR 2a | LOE B-NR)

What it adds

- BMI and eGFR sit in the base equations; uACR, A1C, and social deprivation index are add-ons
- One set of inputs returns ASCVD, HF, and total CVD risk
- Available at professional.heart.org and increasingly built into EHR risk modules

Three uses, and they are different

- Staging — PREVENT-CVD ≥ 20% can place a patient at CKM stage 3
- HF risk — PREVENT-HF ≥ 5% is the threshold for evaluating pre-HF
- Therapy prioritization — PREVENT-CVD ≥ 7.5% helps prioritize SGLT2i and/or GLP-1–based therapy when otherwise indicated



Available at professional.heart.org

Staging characterizes where the patient is. PREVENT quantifies their risk.

EHR = electronic health record; GLP-1: glucagon-like peptide-1; LOE B-NR = Level of Evidence B–Nonrandomized; SGLT2i = sodium-glucose cotransporter 2 inhibitor.

PREVENT calculator inputs per the American Heart Association [AHA]. professional.heart.org. Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Audience Response



A 58-year-old with obesity and hypertension has an eGFR of 88 mL/min/1.73 m^2, a uACR of 55 mg/g, and no known CVD. Which interpretation best reflects the CKM framework?

A. Preserved eGFR suggests that kidney and cardiovascular risk can be assessed independently despite albuminuria

B. Albuminuria indicates kidney involvement, but integrated cardiovascular risk assessment is unnecessary while eGFR remains above 60 mL/min/1.73 m^2

C. Albuminuria despite preserved eGFR signals kidney involvement within interconnected CKM risk and should prompt integrated risk assessment

D. Cardiovascular risk assessment should be deferred until symptoms develop or clinical cardiovascular disease becomes apparent

E. I don’t know

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ARS POLLING RESULTS

Placeholder



Three Things to Carry Forward

1 **This is one process with several presentations.**
Adiposity upstream; insulin resistance, inflammation, endothelial dysfunction, and neurohormonal activation – including RAAS and aldosterone/mineralocorticoid receptor signaling – downstream; the overlap between conditions is where risk concentrates.

2 **Staging turns that process into something you can do at the desk.**
And it identifies where the window for prevention is still open.

3 **PREVENT adds the quantitative half.**
Where the patient is, and how fast they are moving, used together.



Next: finding these patients before any of it is symptomatic.

RAAS = renin-angiotensin-aldosterone system.

CME OUTFITTERS

Identifying Risk Earlier: Practical Screening and Risk Stratification

Lucia M. Novak, MSN, CRNP, ANP-BC, BC-ADM, FAANP



Audience Response



Thinking about kidney testing in your own practice, where does the process most often break down?

A. Ordering: the uACR does not get ordered in the first place

B. Interpreting: the result comes back and it is not clear what it means

C. Acting: we see the result, but nothing in the plan changes

D. Following up: it gets done once and never repeated

CME OUTFITTERS

ARS POLLING RESULTS

Placeholder



uACR: The Cardiac Test Hiding in the Urine Cup

We order it to stage kidney disease. Its stronger signal may be cardiovascular.

- Albuminuria is an independent marker of cardiovascular risk, not only a marker of kidney injury
- This is an argument for ordering it — not for treating it as a nephrology-only test

The recommendation, verbatim:

Among adults in CKM stages 2 and above, assessments of both eGFR and uACR are recommended at least annually to characterize risk related to CKD and to guide the prevention and management of CVD and kidney disease [COR 1 | LOE B-NR]

Why neither test alone is sufficient:

- eGFR – assess function
- uACR – assess structure
- Need **both** to properly stage CKD
- CKD is diagnosed by **either** chronic eGFR < 60 mL/min/1.73 m² **or** uACR ≥ 30 mg/g; an eGFR-only strategy misses albuminuric patients with preserved filtration

Who is stage 2?

Hypertension, T2D, metabolic syndrome, triglycerides ≥ 150 mg/dL, or moderate- to high-risk CKD

A small test. A big picture.



- **Simple**
- **Widely available**
- **High-impact information**

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Reading the Grid: eGFR x Albuminuria

Orient to the heat map first

Rows = eGFR category (G) **Columns = albuminuria category (A)** **The cell = combined CKD risk**

eGFR mL/min/1.73 m²	A1 uACR < 30	A2 30 - < 300	A3 ≥ 300
G1 ≥ 90	Low (if no other markers of kidney disease, not CKD)	Mod	High
G2 60-89	Low (if no other markers of kidney disease, not CKD)	Mod	High
G3a 45-59	Mod	High	Very high
G3b 30-44	High	Very high	Very high
G4 15-29	Very high	Very high	Very high
G5 < 15 or on dialysis	Very high	Very high	Very high

KDIGO CKD risk grid: use the row + column intersection, not either number alone.

Low | Moderate | High | Very high

Then apply the patient cell

Moderate-to-high-risk CKD defines CKM stage 2

G1-G2 with A2-A3; G3a with A1-A2; G3b with A1

Very high-risk CKD is a stage 3 risk equivalent

G3a with A3; G3b with A2-A3; G4-G5

Patient example
eGFR 95 + uACR 60 mg/g
= G1A2 → CKM stage 2

Orient → find the cell → apply to the patient

Heat map colors indicate risk, not class of recommendation.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Adapted from Kidney Disease: Improving Global Outcomes [KDIGO]. *Kidney Int*. 2024;105(4S):S117-S314.

Getting the Numbers Right

uACR

Timing and amount
- First-morning void is preferred
- Do not make time of day a barrier; spot urine is acceptable; 24-hour collection is not required

Confirm persistence
- Repeat an abnormal result before calling it persistent albuminuria

Check for transient elevations
- Fever, heavy exercise in prior 24 hours, UTI, marked hyperglycemia, decompensated HF, menstruation

Interpret change in context
- A doubling of uACR exceeds expected biological and analytic variability and warrants evaluation/confirmation

Dipstick is not enough
- Protein dipstick misses the A2 range that changes management

eGFR

Trend rather than snapshot
- One value tells you far less than three over 18 months

Know what exceeds expected variability
- A > 20% change in eGFR exceeds expected biological and analytic variability and warrants evaluation

Expect the early hemodynamic dip
- A ≤ 30% dip after starting a RAAS inhibitor, MRA, or SGLT2i can be expected

Dipstick is not enough
- Protein dipstick misses the A2 range that changes management

Practical takeaway: confirm the albuminuria signal, trend the filtration signal, and do not stop guideline-directed therapy for an expected early eGFR dip.

UTI = urinary tract infection.

Adapted from KDIGO. *Kidney Int*. 2024;105(4S):S117-S314. Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

How Often, By Stage

ALL — **Every adult, regardless of stage**

- BMI and waist circumference at least annually
- BP at least annually [COR 1 | LOE B-NR]

0 — **Stage 0**
No CKM risk factors

- Lipids, glycemia, and eGFR at least every 5 years

1 — **Stage 1**
Excess or dysfunctional adiposity

- Lipids, glycemia, and eGFR at least every 2 to 3 years

2+ — **Stage 2 and above**
Metabolic risk factors, CKD, subclinical or clinical CVD

- Lipids, glycemia, and BP at least annually
- eGFR *and* uACR at least annually

✓ **All five of these recommendations are COR 1, LOE B-NR.**

Natriuretic Peptides: When They Help, When They Mislead

Reach for one when:

Lower-extremity edema in a patient who already has albuminuria
- A practical trigger worth teaching directly

New or worsening exertional dyspnea, fatigue, or reduced exercise tolerance
- In a patient with T2D, CKD, or obesity

PREVENT-HF 10-year risk ≥ 5%
- As a pre-HF case-finding step (COR 2a | LOE B-NR)

In STOP-HF
- BNP testing with reflex echocardiography and coordinated referral at BNP ≥ 50 pg/mL was associated with roughly 45% lower odds of developing LV dysfunction or HF

Interpret carefully when:

Older age, atrial fibrillation, or CKD
- Each can raise natriuretic peptide levels and lower the specificity of standard cut points

Obesity
- Levels can be lower despite real cardiac dysfunction; if symptoms are significant, assess clinically and consider cardiology/urgent evaluation based on severity

Advanced CKD or established ASCVD
- Elevated levels are common, and incremental value is often limited

Know the pre-HF thresholds (for screening/case finding)
- BNP ≥ 35 pg/mL
- NT-proBNP ≥ 125 pg/mL

Natriuretic peptides are most useful as part of the clinical picture. They can support earlier recognition of possible HF but should not be used for routine monitoring in asymptomatic patients. BNP or NT-proBNP? Either can support HF evaluation. Prefer NT-proBNP in patients taking sacubitril/valsartan because neprilysin inhibition can increase BNP. Kidney dysfunction can elevate both (particularly NT-proBNP), so interpret in clinical context.

CME OUTFITTERS

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Ledwidge M, et al. *JAMA*. 2013;310(1):66-74.

Patient Case 1



64-year-old Black woman with hypertension and T2D

- BMI: 31 kg/m^2
- Reports new exertional dyspnea when climbing stairs

Laboratory trend:

- 12 months ago: eGFR 78 mL/min/1.73 m^2
- 6 months ago: eGFR 68 mL/min/1.73 m^2
- Today: eGFR 62 mL/min/1.73 m^2
- uACR: 45 mg/g

Which interpretation is most appropriate?

CME OUTFITTERS

Audience Response

A 64-year-old Black woman with hypertension, T2D, and a BMI of 31 kg/m^2 has an eGFR decline from 78 → 68 → 62 mL/min/1.73 m^2 over 12 months, uACR 45 mg/g, and new exertional dyspnea on stairs. Which interpretation is most appropriate?

A. Her eGFR remains above 60, making CKD unlikely; the new dyspnea is probably unrelated to kidney risk

B. She has G2A2 CKD with declining eGFR, and the new dyspnea warrants evaluation for heart failure

C. Her preserved eGFR and moderately increased albuminuria indicate low kidney risk; cardiovascular risk remains unchanged

D. She has G2A2 CKD, but the dyspnea is most consistent with deconditioning associated with excess weight

E. I don't know

G2A2 = KDIGO GFR category 2, albuminuria category 2.

CME OUTFITTERS

ARS POLLING RESULTS

Placeholder



Case 1: Discussion

1	**Stage her**	G2A2 by KDIGO is moderate-risk CKD. With T2D and hypertension, she is CKM stage 2 today at this visit.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Inker LA, et al. *N Engl J Med*. 2021;385(19):1737-1749.

Case 1: Discussion

1	**Stage her**	G2A2 by KDIGO is moderate-risk CKD. With T2D and hypertension, she is CKM stage 2 today at this visit.
2	**Read the trend, not the number**	A fall from 78 to 62 is roughly 20% in 12 months, which exceeds expected variability. The 62 is not reassuring; the slope is the finding. Confirm the lab uses the race-free 2021 CKD-EPI equation; race-based equations ran higher in Black patients and delayed recognition.

CKD-EPI = Chronic Kidney Disease Epidemiology Collaboration.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Inker LA, et al. *N Engl J Med*. 2021;385(19):1737-1749.

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3	**Confirm the albuminuria**	Repeat the uACR to establish persistence before building a plan on it.

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RASi = renin-angiotensin system inhibitor.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Inker LA, et al. *N Engl J Med*. 2021;385(19):1737-1749.

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5	**Take the dyspnea seriously**	New exertional dyspnea in a patient with T2D and CKD warrants evaluation for HF rather than attribution to BMI or deconditioning. Natriuretic peptides support the evaluation, but an elevated level alone does not confirm HF; echocardiography is needed since CKD can ↑ and obesity can ↓ levels.

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6	**Before she leaves:**	Repeat uACR, BNP or NT-proBNP as clinically appropriate, and measure follow-up in weeks rather than a year. Ensure she is on max tolerated dose of RAAS inhibitor. Add SGLT2i (and other measures, as appropriate). Refer to cardiology. **See next slide.**

CME OUTFITTERS

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. Inker LA, et al. *N Engl J Med*. 2021;385(19):1737-1749.

You Found CKM Syndrome. What Can You Do Before Referral?

Start guideline-directed treatment in primary care. Refer when indicated, but do not wait to begin appropriate care.

K Chronic Kidney Disease (CKD)

Slow progression. Reduce cardiovascular risk.

Foundational therapy

SGLT2 inhibitor
Use if eGFR ≥ 20 mL/min/1.73 m²
Slows CKD progression; reduces HF/CV events

ACEi or ARB
Use for hypertension with CKD/albuminuria, and other established cardiovascular indications, unless contraindicated

Add by phenotype/residual risk

Finerenone/nsMRA
T2D + persistent albuminuria despite max tolerated RASi
Monitor potassium and eGFR

GLP-1 RA
T2D and/or obesity
CV benefit; kidney-protective benefit in appropriate patients with T2D

Risk-factor control
Blood pressure, lipids, glycemia, weight management, lifestyle

These therapies are within reach of the PCP. Start appropriate treatment now; refer or co-manage when indicated.

HF HFpEF

Relieve symptoms. Reduce hospitalizations. Treat comorbidities.

Evidence-based therapies within PCP reach

SGLT2 inhibitor with HF indication
Recommended for HFpEF, with or without diabetes; reduces HF hospitalization/CV events

Loop diuretic
Use for congestion/volume overload; titrate to symptoms, exam, and weight

MRA
Consider in selected patients; monitor potassium and renal function

GLP-1-based therapy
Consider in HFpEF with obesity to improve symptoms and QoL and reduce HF events. (Note: no GLP-1-based therapy currently carries a U.S. HF indication.)

When to refer/co-manage: eGFR < 30 | rapid unexplained eGFR decline | uACR ≥ 300 mg/g | resistant hypertension | recurrent HF hospitalizations or persistent symptoms | uncertain diagnosis/etiology

ACEi = ACE inhibitor; QoL = quality of life.
Not all agents in a class carry the same indication. See appendix handout for agent-level indications.
Adapted from Ndumele CE, et al. *J Am Coll Cardiol.* 2026;87(22S):e1889-e2007. KDIGO. *Kidney Int.* 2024;105(4S):S117-S314.

CME OUTFITTERS

Do Something: The Family Medicine Action Pathway

A practical workflow for earlier identification and management of CKM across the care continuum



Identify CKM risk or overlapping disease

- T2D
- Hypertension
- Obesity
- CKD
- HF symptoms (e.g., dyspnea, edema, fatigue)

Order the right tests

- eGFR + uACR (for CKD)
- Add BNP or NT-proBNP when HF is suspected

Determine risk and stage

- Use CKM stage and KDIGO risk category
- Consider PREVENT for ASCVD risk where appropriate

Begin appropriate therapy

- Initiate indicated kidney- and CV-protective therapy

Do not wait for referral!

Track response and safety

- Trend eGFR, uACR, potassium, BP, and symptoms
- **Expect and interpret treatment-related changes appropriately**

Escalate when indicated

- High-risk features (e.g., eGFR < 30, uACR ≥ 300 mg/g)
- Diagnostic uncertainty
- Persistent symptoms despite treatment
- Continue treatment while referral is pending

The primary care action is not "identify and refer."
It is identify, act, monitor, and co-manage.

CME OUTFITTERS

Ndumele CE, et al. *J Am Coll Cardiol.* 2026;87(22S):e1889-e2007. KDIGO. *Kidney Int.* 2024;105(4S):S117-S314.

Applying Guideline-Aligned, Patient-Centered Management

Jay H. Shubrook, DO, FAAFP, FACOFP



Three Guidelines, One Patient, One Plan

ADA | KDIGO | ACC/AHA/HFSA

The reconciliation problem is real.

ADA, KDIGO, and ACC/AHA/HFSA each publish a defensible algorithm for the same patient, organized around different diseases.

Where they already agree – and the agreement is most of it:

- **Screen with both eGFR and uACR** in anyone with diabetes, hypertension, or CVD
- **RAAS inhibition** at maximally tolerated dose for CKD with albuminuria
- **SGLT2 inhibition** as foundational cardiorenal therapy rather than a glucose drug
- **Escalate on persistent albuminuria** rather than accepting it
- **BP target < 130/80 mm Hg; A1C < 7.0%** for most nonpregnant adults without significant hypoglycemia

The 2026 CKM guideline issues no recommendations of its own for blood pressure or lipids, deferring to the 2025 blood pressure and 2026 dyslipidemia guidelines.

mm Hg = millimeters mercury.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

CKD + T2D: What Can You Start, and What Comes Next?

Use guideline-directed therapy to reduce kidney and CV risk. Start now, reassess, and add therapy as indicated.

1 START
RASi + SGLT2i as indicated

RASi
- ACEi or ARB
- Use in hypertension, diabetes, or albuminuria unless contraindicated
- Titrate to maximally tolerated dose

SGLT2i
- Use if eGFR ≥ 20 mL/min/1.73 m²
- Slows CKD progression
- Reduces CV events

2 REASSESS uACR
Persistent albuminuria?

Check uACR and eGFR

Reassess after initiation and dose optimization (e.g., **3-6 months**)

Persistent albuminuria?
(e.g., uACR ≥ 30 mg/g)

No
Continue current therapy and monitor

Yes
Consider adding nsMRA and/or GLP-1 RA

3 ADD
nsMRA and/or GLP-1–based therapy as indicated

nsMRA (finerenone)
- T2D + persistent albuminuria despite maximally tolerated RASi
- Monitor potassium and eGFR
- Reduces CKD progression and CV events

GLP-1 RA
- Consider for T2D, especially with obesity or additional CV risk
- Reduces CV events
- Kidney-protective benefit in appropriate patients with T2D

These therapies are within reach in family medicine.

Start appropriate treatment now, reassess, and add therapy as indicated. Refer or co-manage for high-risk features or diagnostic uncertainty. Not all agents in a class carry the same indication. See appendix handout for agent-level indications.

nsMRA = nonsteroidal mineralocorticoid receptor antagonist.

Adapted from Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007. KDIGO. *Kidney Int*. 2024;105(4S):S117-S314.

How Fast Should You Build the Regimen?

The instinct is to add one drug, wait, reassess, add another.

The evidence increasingly supports moving faster.

Simultaneous versus sequential: what CONFIDENCE showed (Phase II; albuminuria endpoint at day 180)

- 818 adults with CKD and T2D; finerenone and empagliflozin started together versus either alone
- Median uACR fell 52% from baseline at day 180 with combination therapy
- 29% greater reduction than finerenone alone, and 32% greater than empagliflozin alone
- More than 30% reduction was evident by day 14
- Hyperkalemia: 15.3% with combination, 18.6% with finerenone alone, 9.7% with empagliflozin alone

Why this matters more in primary care than in the clinic that ran the trial

- Every deferred decision is a visit away, and if the patient is also waiting on a specialist appointment, sequential initiation can stretch across a year.
- These are approved agents with guideline recommendations. Combining them earlier is within, not ahead of, current practice. Named agents reflect the trial protocol; the principle is class-level, a nonsteroidal MRA plus an SGLT2i on maximally tolerated RAS inhibition.

The realistic caution is monitoring capacity, and the number you are watching is potassium.

If you can check an eGFR and a potassium in 2 to 4 weeks, you can start more than one thing.

CME OUTFITTERS

Agarwal R, et al. *N Engl J Med*. 2025;393(6):533-543. Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

CKD Without T2D – and the Overlaps

CKD Without T2D

uACR ≥ 30 mg/g eGFR ≥ 30	RAASi at maximum tolerated dose **COR 1 | LOE B-R**
uACR ≥ 200 mg/g eGFR ≥ 20	SGLT2i **COR 1 | LOE A**
uACR 30–199 mg/g	SGLT2i can be considered **COR 2a | LOE B-R**

The evidence is strongest at higher albuminuria.

Common Overlaps

Obesity + T2D, PREVENT-CVD ≥ 7.5%	SGLT2i or GLP-1-based therapy **COR 1 | LOE A**
Obesity + symptomatic HFpEF	GLP-1-based therapy with proven CV benefit **COR 1 | LOE A**
	Guideline-supported for obesity-related symptomatic HFpEF; not an FDA-approved HF indication.
Pre-HF (stage 3) with T2D or CKD	SGLT2i first-line for HF prevention **COR 1 | LOE B-NR**
T2D or ≥ 2 cardiometabolic risk factors	FIB-4 every 1 to 2 years to screen for MASLD-related fibrosis **COR 1 | LOE B-NR**

Not all agents in a class carry the same indication. See appendix handout for agent-level indications.
FIB-4 = fibrosis-4.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Patient Case 2



66-year-old Indigenous man with T2D

- HF hospitalization one year ago
- Current medications: metformin, an ACEi, and a loop diuretic

Test results:

- A1C: 8.2%
- eGFR: 58 mL/min/1.73 m^2
- uACR: 160 mg/g
- BP: 138/82 mm Hg

Which management approach best reflects guideline-aligned, integrated CKM care?

CME OUTFITTERS

Audience Response



A 66-year-old with T2D (A1C 8.2%), eGFR 58, uACR 160 mg/g, BP 138/82, and an HF hospitalization one year ago is taking metformin, an ACEi, and a loop diuretic. Which management approach best reflects guideline-aligned, integrated CKM care?

A. Intensify glycemic therapy first and reassess kidney and HF risk after A1C improves

B. Add an SGLT2i, optimize ACEi therapy, and reassess kidney risk and albuminuria

C. Increase loop diuretic therapy and reassess symptoms and kidney function before adding other therapy

D. Refer to nephrology and cardiology and defer medication changes until specialty evaluation

E. I don’t know

CME OUTFITTERS

ARS POLLING RESULTS

Placeholder



Case 2: What Can You Initiate or Optimize Today?

A practical plan for this patient in the primary care setting

1 INITIATE/OPTIMIZE NOW

Evidence-based therapy within your scope of practice

Rx — ACEi

Confirm maximally tolerated dose

Rx — SGLT2i

Initiate now for kidney, CV, and HF benefit

Glucose-lowering efficacy declines as eGFR falls below ~60; cardiorenal benefit persists.

✓ — Continue metformin

Maintain glycemic therapy; do not rely on SGLT2i alone for A1C control

2 PLANT THE NEXT DECISION

Reassess and escalate as indicated

Lab — Reassess uACR

in 3-6 months

↗ — If uACR remains ≥ 30 mg/g

despite maximally tolerated ACEi + SGLT2i, and eGFR is ≥ 25 mL/min/1.73 m², consider adding a nsMRA

3 SCHEDULE MONITORING

Before the patient leaves

K+ — eGFR + potassium

2-4 weeks

u — uACR

3-6 months

A1C — A1C

3-6 months

Do not wait for specialty consultation to begin indicated therapy; initiate, monitor, and escalate based on response.

CME OUTFITTERS

Ndumele CE, et al. *J Am Coll Cardiol.* 2026;87(22S):e1889-e2007.

Monitoring Is What Makes Moving Quickly Safe

After starting or escalating

RAASi or MRA

Recalculate eGFR and measure potassium at 2 to 4 weeks
COR 2a | LOE B-R

Kidney-protective therapy with uACR ≥ 30

Remeasure albuminuria at 3 to 6 months to assess residual risk
COR 2a | LOE B-R

Cardioprotective glucose-lowering agent

Assess glycemia every 3 to 6 months – sooner if A1C is above target
COR 1 | LOE A

GLP-1–based therapy for weight

Reassess at 3 to 6 months; under 5% weight loss should prompt dose escalation, a switch, or referral
COR 1 | LOE B-NR

Expected versus a real problem

EXPECTED – KEEP GOING

- An early eGFR decline up to ~30% may be hemodynamic and does not automatically require discontinuation; assess volume status, potassium, and other causes.
- Potassium ≤ 5.5 mEq/L: often tolerable with dietary review and monitoring
- eGFR later falls below a drug's initiation threshold: reasonable to continue as safely tolerated **COR 2a | LOE B-R**

A REAL PROBLEM – ACT ON IT

- eGFR falls more than 30%: look for another cause before abandoning therapy
- Potassium > 5.5 mEq/L: needs mitigation. SGLT2is themselves reduce the incidence of hyperkalemia during RAAS inhibition.

Protect the kidney and treat the glucose—not one or the other.
SGLT2is move A1C only modestly, and hyperglycemia is itself nephrotoxic. If A1C stays above target, add or escalate an agent that keeps the CKD and HF benefit (semaglutide or another GLP-1 receptor agonist) and recheck A1C in 3 months.

SGLT2i safety: assess volume status; counsel on genital infection and ketoacidosis risk. Hold during critical illness or prolonged fasting and before scheduled surgery (generally 3-4 days).
LOE B-R = Level of Evidence B–Randomized.

Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Who Owns the CKM Plan Between Visits?

CKM is a team effort, and the primary care clinician leads.

PCP

PRIMARY CLINICIAN/CARE LEAD
Initiates, adjusts, and owns the CKM plan

May be:

- Family physician (MD/DO)
- NP
- PA

TEAM

CORE TEAM SUPPORT
Ongoing management + patient support

May include:

- Clinical pharmacist
- Nurse
- Diabetes care and education specialist
- Other team members based on patient needs

WHAT THE TEAM NEEDS TO ACCOMPLISH

Comprehensive medication management	Lifestyle support	Care coordination	Patient navigation	Screening for and addressing relevant social needs

Referral is not the same as coordination.
Someone on the primary care team should own the plan between visits.

MD = doctor of medicine; DO = doctor of osteopathic medicine; NP = nurse practitioner; PA = physician associate.

Adapted from Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

Patient Case 3



59-year-old Black woman with T2D and early CKD

- Reports difficulty attending frequent appointments because of work constraints

Test results:

- eGFR: 70 mL/min/1.73 m^2
- uACR: 110 mg/g

Where would you start with this patient knowing there are multiple barriers?

CME OUTFITTERS

Audience Response



A 59-year-old with T2D and early CKD (eGFR 70, uACR 110 mg/g) reports difficulty attending frequent appointments because of work constraints. Which plan best balances guideline alignment and patient capacity?

A. Defer therapy changes until she can commit to the standard monitoring schedule

B. Start all indicated therapies now and monitor as her schedule permits

C. Start the highest-yield agent now, consolidate lab draws, and set a date to reassess escalation

D. Refer to a specialist and defer treatment decisions until consultation

E. I don’t know

CME OUTFITTERS

ARS POLLING RESULTS

Placeholder



Shared Decision-Making That Fits a 15-Minute Visit

The Three-Talk Model

Team talk

"We have some choices to make together. Let me tell you what they are."

Option talk

Describe the realistic options and what each one asks of her, not only what it does for her.

Decision talk

"What matters most to you here?" Then decide and name the decision out loud.

Minimally Disruptive Medicine

Treatment burden is a clinical variable. Every added drug, lab, and visit spends a finite amount of her capacity.

Practical moves for this patient:

- Consolidate lab draws with visits she is already making
- Choose the highest-yield agent first rather than stacking
- Raise the cost question before she has to ask it twice
- Set an explicit date for the next decision, so "not now" does not become "never"
- Use virtual follow-up when appropriate to reduce time off work

A regimen she cannot sustain is not a more aggressive plan. It is a shorter one.

Adapted from Elwyn G, et al. *BMJ*. 2017;359:j4891. May C, et al. *BMJ*. 2009;339:b2803. Ndumele CE, et al. *J Am Coll Cardiol*. 2026;87(22S):e1889-e2007.

CME OUTFITTERS



Put information into action!

Takeaways from this program can be implemented into your practice to improve patient care.

- Over the next 90 days, order eGFR and uACR together for every patient at CKM stage 2 or above who does not have both on file from the past 12 months
- Stage every patient with T2D seen in the next 90 days using the CKM framework, document the stage, and set the screening interval that stage calls for (every 5 years at stage 0, every 2 to 3 years at stage 1, and annually at stage 2 and above)
- In the next 90 days, start guideline-directed cardiorenal therapy at the visit where the indication is identified, rather than waiting for a specialist consultation
- Repeat the uACR 3 to 6 months after starting or escalating kidney-protective therapy, rather than waiting for the annual visit
- Within 30 days, identify who in my practice will own the 2-to-4-week eGFR and potassium check after a RAAS inhibitor or MRA is started

QUESTIONS & ANSWERS



Additional Resources

Visit www.cmeoutfitters.com for clinical information and certified educational activities

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CME OUTFITTERS

Improving Early Identification and Integrated Management

of Cardio-Renal-Metabolic Risk in Family Medicine



This activity is supported by an independent medical educational grant from Boehringer Ingelheim Pharmaceuticals, Inc.