



Cardiovascular-Kidney-Metabolic (CKM) Syndrome

2026 AHA/ACC/ADA/ASN Guideline (JACC, June 2026) · retires the 2013 AHA/ACC/TOS obesity guideline

Stage decides what & how often

THE FIVE STAGES *bidirectional: the point is regression, not just prevention*

Stage	Definition	What it changes for you
0	No CKM risk factors. Normal weight, glucose, BP, lipids and kidney function, no subclinical or clinical CVD	Goal is keeping them here. Widest screening interval; lifestyle counseling is the whole intervention
1	Excess or dysfunctional adiposity: overweight, obesity, or abdominal obesity, may include prediabetes. Without Stage 2 metabolic risk factors and without subclinical CVD	First actionable stage and the most often ignored, because the patient feels well . Weight loss here can drive true stage regression
2	Metabolic risk factors or moderate-to-high-risk CKD: hypertension, T2DM, hypertriglyceridemia, metabolic syndrome, or CKD in moderate-to-high-risk KDIGO categories	Screening tightens sharply (annual lipids, glycemia, BP, and eGFR <i>and</i> UACR). Entry point for cardioprotective drug therapy
3	Subclinical CVD or risk equivalent: CAC > 100, pre-HF (NT-proBNP ≥ 125 pg/mL with echo dysfunction, or elevated hs-troponin), very high-risk CKD , or PREVENT 10-yr CVD risk ≥ 20%	Last stop before clinical disease. In pre-HF Stage 3, intensive lifestyle plus intensive risk factor control is Class 1 , because clinical HF is still preventable
4	Clinical CVD (CHD, HF, stroke, PAD, atrial fibrillation) overlapping CKM risk factors. 4a without kidney failure, 4b with kidney failure	Treating the coronary disease while ignoring obesity, diabetes and albuminuria leaves most of the modifiable risk on the table

Stage youths under 18 as well as adults. The stages are explicitly bidirectional, which is the practical difference from a risk calculator: a score gives a probability and implies nothing can be undone, while the stage tells you what to try to reverse.

THE FOUR CKD THRESHOLDS *written in mg/g and mL/min, and easy to mix up*

Agent	Who qualifies	Why / what it prevents
RAS inhibitor (ACEi or ARB) Class 1	CKD with T2DM , or without T2DM but UACR ≥ 30 , and eGFR ≥ 30	Lowers intraglomerular pressure and slows GFR loss. Maximum tolerated dose is the recommendation , not a token dose, and that is the step most often left undone
SGLT2 inhibitor Class 1	CKD with T2DM , or without T2DM but UACR ≥ 200 , and eGFR ≥ 20	Slows GFR loss, cuts HF hospitalization and CV mortality (DAPA-CKD, EMPA-KIDNEY). Expect an early eGFR dip and do not stop for it: it is hemodynamic and precedes the benefit
SGLT2 inhibitor Class 2a	CKD without T2DM , UACR 30 to 199	Fills the gap below the Class 1 albuminuria threshold, where benefit is likely but the trial evidence is less direct
Finerenone (nonsteroidal MRA) Class 1	CKD + T2DM + UACR ≥ 30 despite ACEi/ARB and SGLT2i as tolerated, with eGFR ≥ 25	Targets the residual albuminuria that persists on standard therapy (FIDELIO-DKD). Added <i>despite</i> the other two, not instead of them. Monitor potassium

You cannot apply any of these without a UACR, because every threshold is written in mg/g. eGFR alone misses early kidney disease: albuminuria appears first and independently predicts both kidney and cardiovascular events.

TYPE 2 DIABETES *drug choice follows risk, not A1c*

Recommendation	Threshold	Why
SGLT2i or GLP-1-based therapy with proven benefit Class 1	Stage 2 to 3 with T2DM and PREVENT 10-yr CVD ≥ 7.5%	Benefit is largely independent of glucose lowering , which is why the trigger is cardiovascular risk rather than A1c
Intensified multifactorial treatment Class 1	Stage 2 to 3 with T2DM	Treating glycemia, BP, lipids and albuminuria as a bundle beats sequential single-target care for CVD, mortality and kidney outcomes
Metformin, added to a cardioprotective agent Class 2a	A1c 0.5% to 1% above the individualized goal	Note the reversal: metformin is now what you add for glycemic control <i>after</i> the cardioprotective drug, not the mandatory first step before it
GLP-1 plus SGLT2i together Class 2b	Increased CVD risk or multiple CKM risk factors	May add benefit beyond either alone; the weak class reflects thin dedicated combination outcome data

SCREENING, SET BY STAGE *and the two numbers that replace BMI alone*

EVERY ADULT BMI and waist circumference, both. Obesity at BMI ≥ 30; abdominal obesity at ≥ 88 cm in women, ≥ 102 cm in men . BMI alone misclassifies: it misses the normal-BMI patient with central adiposity and overcalls the muscular one.	FROM STAGE 2, ANNUALLY Lipids, glycemia, blood pressure, and eGFR with UACR , all Class 1. PREVENT risk in Stages 0 to 3: ≥ 20% 10-year risk is itself a Stage 3 criterion , and ≥ 7.5% prioritizes starting cardioprotective therapy.	DO NOT FORGET FIB-4 every 1 to 2 years with diabetes or ≥ 2 risk factors: MASLD travels with this syndrome, is silent, and FIB-4 costs nothing extra. Pre-HF biomarkers (BNP/NT-proBNP, hs-troponin) if PREVENT-HF ≥ 5%, Class 2a. Screen for OSA .
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WHAT CHANGED, AND WHAT GETS MISSED

- Obesity is the entry point, and the conversation is the intervention.** Lifestyle first-line targeting **5% to 10%** of baseline weight, and counsel about that benefit **at least annually** (Class 1). That range is where blood pressure, glycemia, triglycerides and hepatic steatosis reliably improve **even if the patient stays in the obese BMI range**.
 - Two reversals worth knowing:** metformin has moved from mandatory first step to a Class 2a glycemic add-on, and **BMI alone is no longer adequate** without a waist circumference.
- Social determinants are clinical data, not paperwork.** Food insecurity, housing instability and cost barriers predict both the development of CKM syndrome and the failure of the plan you are writing. **A GLP-1 the patient cannot afford is a failed prescription.**
 - Add finerenone despite the other two, not instead.** The commonest error is treating the four thresholds as a ladder where each replaces the last, when RAS inhibitor, SGLT2i and finerenone are meant to stack.