

STAGE IT ON TWO AXES *albuminuria is not optional — it drives prognosis and therapy*G STAGE — eGFR (mL/min/1.73 m²)

G1 ≥ 90 · **G2** 60–89 · **G3a** 45–59 · **G3b** 30–44 · **G4** 15–29 · **G5** < 15. CKD requires abnormality present for > **3 months**. A single low eGFR is not CKD.

A STAGE — urine albumin-to-creatinine ratio (mg/g)

A1 < 30 · **A2** 30–300 · **A3** > 300. A patient with a normal eGFR and A3 albuminuria has CKD and substantial risk. **Check a UACR in everyone you stage.**

THE PILLARS OF KIDNEY PROTECTION *this is where progression is actually slowed*

Therapy	Who qualifies	Notes
RAS inhibitor ACEi or ARB	CKD with albuminuria , especially A2–A3, with or without diabetes	Titrate to the maximum tolerated dose. Do not stop for a stable creatinine rise of up to ~30% — that reflects intraglomerular pressure reduction, which is the point.
SGLT2 inhibitor	eGFR ≥ 20 with UACR ≥ 200 mg/g ; or T2D + CKD with eGFR ≥ 20 ; or heart failure at any albuminuria	Roughly 37% reduction in kidney disease progression and 23% reduction in AKI, irrespective of diabetes status . Expect a small initial eGFR dip — continue through it.
Finerenone non-steroidal MRA	T2D, eGFR > 25, normal potassium , and albuminuria > 30 mg/g despite a maximally tolerated RAS inhibitor	Requires careful monitoring of potassium and eGFR , with dose reduction or discontinuation if needed. Adds benefit on top of RASi and SGLT2i.
Blood pressure	Most adults with CKD	Target office BP < 130/80 mmHg , using standardised measurement technique.
Statin	Most adults with CKD	CKD is a cardiovascular risk state. Most patients with CKD die of cardiovascular disease, not of dialysis.

MANAGE THE COMPLICATIONS *each has a threshold worth remembering*

ANEMIA

Replete iron first — target ferritin and transferrin saturation before starting an erythropoiesis-stimulating agent. **Do not target a normal hemoglobin**; overcorrection increases stroke and thrombosis. Investigate other causes before attributing it to CKD.

MINERAL & BONE

Rising **phosphate** and **PTH** with falling **calcitriol**. Dietary phosphate restriction, then binders — **prefer non-calcium-based**. Treat vitamin D deficiency. Calcimimetics for persistent hyperparathyroidism on dialysis.

ACIDOSIS & POTASSIUM

Oral bicarbonate for serum bicarbonate persistently < 18–22 — slows progression and protects muscle and bone. For **hyperkalemia**, use a potassium binder to **keep the RASi and MRA on board** rather than stopping them.

WHEN TO REFER, AND WHAT TO AVOID *plan the transition long before you need it*

REFER TO NEPHROLOGY

eGFR < 30 (G4–G5) · **UACR > 300** · rapid progression · persistent hematuria with proteinuria · refractory hypertension on 4 agents · recurrent stones · suspected genetic or glomerular disease · persistent electrolyte abnormality. **Early referral allows planned access and modality choice** rather than a crash start on a temporary line.

AVOID / ADJUST

NSAIDs · iodinated contrast where avoidable · nephrotoxic antibiotic combinations such as **vancomycin plus piperacillin-tazobactam** · **metformin below eGFR 30** · renally cleared drugs at unadjusted doses. **Give a sick-day plan** to hold RASi, SGLT2i, diuretics, and metformin during intercurrent illness.

PLANNING FOR KIDNEY FAILURE *start the conversation at G4, not at G5*

Element	What to do, and when
Modality education	Discuss hemodialysis, peritoneal dialysis, transplantation, and conservative care while the patient is still well enough to choose. Conservative management is a legitimate option, particularly in the frail elderly, where dialysis may not extend meaningful life.
Vascular access	Refer for an arteriovenous fistula several months before anticipated need — fistulas take time to mature. Protect the non-dominant arm : no PICC lines, no routine phlebotomy or cannulation.
Transplant workup	Refer early — assessment takes months, and pre-emptive transplantation gives better outcomes than transplanting after years of dialysis. Living donor options should be raised explicitly.
Vaccination	Hepatitis B (higher doses and more doses may be needed in CKD), influenza, pneumococcal, and COVID-19. Do it before immunosuppression or dialysis.
Advance care planning	Document goals and resuscitation preferences. Dialysis withdrawal is a common terminal event and is far easier discussed early than in a crisis.

WHEN THE eGFR DROPS SUDDENLY *AKI on CKD is not just progression*

WORK THROUGH THE REVERSIBLE CAUSES

Volume depletion from over-diuresis or poor intake · **obstruction** — scan the bladder · new **NSAID**, contrast, or nephrotoxic antibiotic · sepsis or hypotension · decompensated heart failure or cirrhosis · rapidly progressive glomerular disease with an active sediment.

THE COMMON MISSTEP

Attributing every rise to "CKD progression" and doing nothing. True CKD progression is gradual over months. A jump over days is an **acute insult** with something reversible behind it — check a urine dipstick, a bladder scan, the drug chart, and the volume status before accepting a new baseline.

PEARLS & PITFALLS

- **A normal eGFR does not exclude CKD.** Isolated A3 albuminuria carries high renal and cardiovascular risk.
- **Do not stop the ACEi for a 25% creatinine rise.** Recheck, look for volume depletion, and preserve the drug that is protecting the kidney.
- **Expect an eGFR dip when starting an SGLT2 inhibitor.** It is hemodynamic and it precedes long-term benefit.
- **SGLT2 inhibitors help regardless of diabetes.** Do not reserve them for diabetic patients.
- **Use a potassium binder to keep the RASi,** rather than sacrificing a mortality-modifying drug.
- **Refer early.** Late referral means a tunnelled line and an emergency start, which carries worse outcomes than a planned fistula.

