NEWSLETTER 6

Routine Dual Kidney Assessment: eGFR + UACR

Making albuminuria testing standard practice to catch CKM early

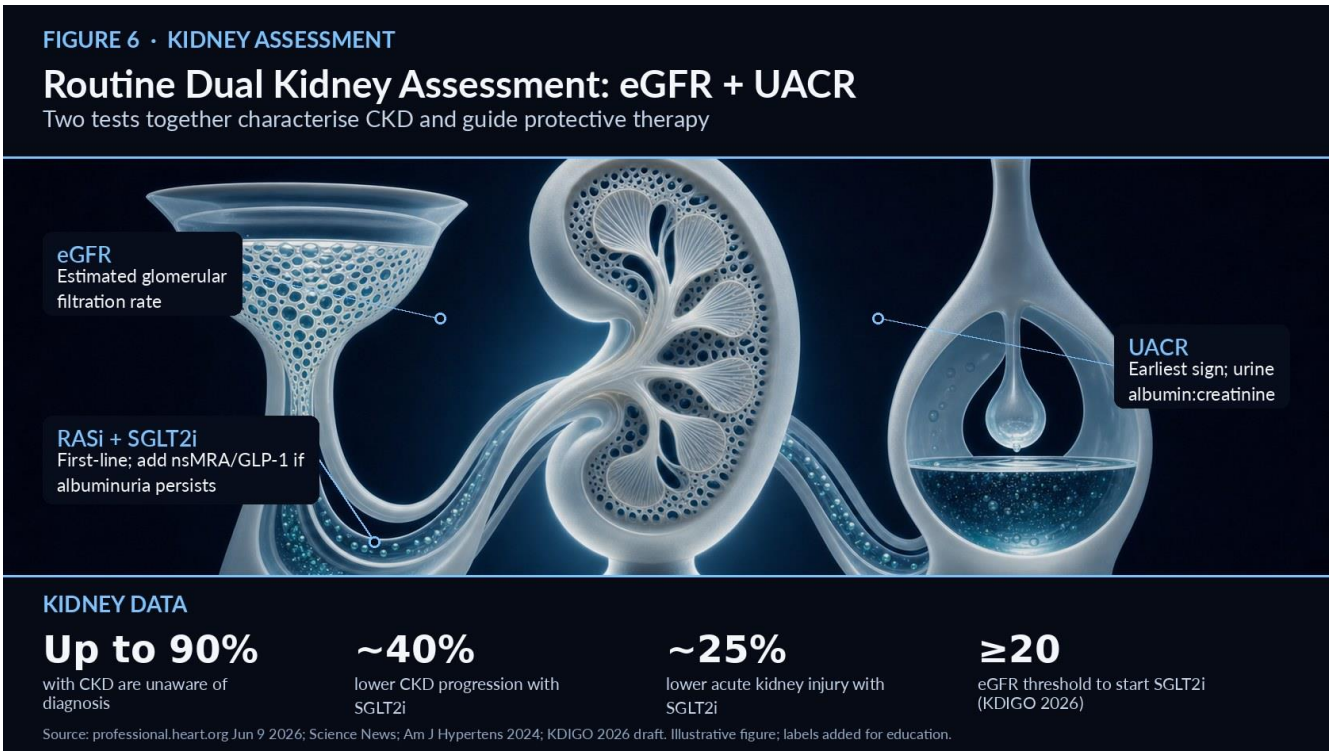

Figure 6. Routine Dual Kidney Assessment: eGFR + UACR — mechanism overview (illustrative; labels added for education).

One of the most operationally consequential recommendations of the 2026 guideline is deceptively simple: use both estimated glomerular filtration rate (eGFR) and urine albumin-to-creatinine ratio (UACR) to characterise chronic kidney disease and to guide therapies that confer both cardiovascular and kidney benefit.[1] Relying on eGFR alone misses a large group of patients whose earliest kidney injury manifests as albuminuria.

The clinical logic is clear. UACR often provides the earliest sign of kidney disease—frequently before filtration-rate testing becomes abnormal—so a normal creatinine can falsely reassure both clinician and patient.[2] This matters enormously given that up to 90% of people with CKD are unaware of their diagnosis, a detection gap that routine dual assessment in primary care is designed to close.[2]

Once CKD is identified, the guideline sets a clear therapeutic sequence: for CKD with type 2 diabetes or with albuminuria, RAS inhibitors and SGLT2 inhibitors should be first-line, and if albuminuria persists, a nonsteroidal MRA or GLP-1-based therapy should be added.[1] Dual assessment thus does more than diagnose—it directly triggers organ-protective treatment.

The evidence base for acting on these numbers is strong. SGLT2 inhibition reduces the risk of acute kidney injury by roughly 25% and slows kidney-disease progression by about 40% on top of standard care in high-risk patients with type 2 diabetes and CKD.[4] The KDIGO 2026 draft reinforces eGFR-based thresholds, recommending SGLT2 inhibitors for adults with type 2 diabetes, CKD, and eGFR ≥20 mL/min/1.73 m² at a 1A strength of recommendation.[5]

The two tests answer different questions and are therefore complementary: eGFR estimates how much filtration capacity remains, while UACR detects glomerular leak and endothelial injury that often precede any fall in filtration.[2] Measuring only one leaves a blind spot. Because the CKM guideline ties kidney-protective therapy directly to these results, a missed UACR is not just a missed diagnosis but a missed opportunity to start RAS inhibitors, SGLT2 inhibitors, and —if albuminuria persists—a nonsteroidal MRA or GLP-1 therapy.[1]

For hospital systems, embedding an automatic UACR order alongside every diabetic or hypertensive creatinine panel is a low-cost, high-yield quality intervention—one that can be tracked as a KPI and that directly advances earlier CKM staging and treatment.[6]

References

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