RESEARCH PAPER

Screening for Early Diabetic Kidney Disease: How the Urinary Albumin-to-Creatinine Ratio (UACR) Performs in Newly Diagnosed Type 2 Diabetes

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ABSTRACT
Among the microvascular complications of type 2 diabetes mellitus (T2DM), diabetic kidney disease (DKD) is one of the most common, and it remains the single biggest driver of chronic kidney disease (CKD) and end-stage kidney disease around the world. Because hyperglycemia can go unnoticed for years, the kidneys are often already damaged by the time T2DM is actually diagnosed — which is exactly why catching DKD early matters, both to slow its progression and to cut the cardiovascular morbidity and mortality that comes with it. The urinary albumin-to-creatinine ratio (UACR) fits that need well: it's a cheap, non-invasive, simple test that's sensitive enough to pick up kidney injury well before estimated glomerular filtration rate (eGFR) starts to fall. That's why current international guidelines call for UACR alongside eGFR at the point of T2DM diagnosis and then yearly after that. Persistent albuminuria doesn't just flag renal disease on the way — it's also an independent marker of cardiovascular risk in its own right. Newer drug classes, among them SGLT2 inhibitors and the non-steroidal mineralocorticoid receptor antagonists, have only reinforced how much catching albuminuria early matters, since starting treatment promptly can meaningfully slow CKD progression. This review pulls together evidence from 2015 to 2026 on UACR's role in newly diagnosed T2DM, walks through the underlying pathophysiology, weighs up current screening guidance, surveys the major clinical studies, and looks ahead at how early detection of DKD might improve further. Current ADA and KDIGO guidance continues to call for combined UACR and eGFR testing from the moment T2DM is diagnosed.

Keywords: Type 2 diabetes mellitus; Diabetic kidney disease; Albuminuria; Urinary albumin-to-creatinine ratio; Chronic kidney disease; Microalbuminuria.
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1. Introduction

Diabetes mellitus (DM) ranks among the fastest-growing non-communicable diseases anywhere, and it's become a genuine public health challenge in its own right. The International Diabetes Federation puts the current global total above 589 million adults living with diabetes, a figure expected to climb past 850 million by 2050 if nothing changes. Type 2 diabetes (T2DM) makes up roughly 90–95% of all cases and carries a heavy load of chronic microvascular and macrovascular complications [1,2].

Diabetic kidney disease (DKD) — once called diabetic nephropathy — is one of the more common microvascular complications tied to T2DM, and worldwide it's still the leading cause behind both chronic kidney disease (CKD) and end-stage kidney disease (ESKD). Somewhere between 30 and 40% of T2DM patients go on to develop DKD, bringing with it higher morbidity, mortality, cardiovascular disease, and healthcare spend [3–5].

What makes this tricky is that kidney damage can already be underway by the time T2DM is formally diagnosed, since hyperglycemia often goes undetected for years beforehand. Sustained high blood glucose sets off glomerular hyperfiltration, endothelial dysfunction, oxidative stress, inflammation, and activation of the renin–angiotensin–aldosterone system (RAAS), all feeding into progressive renal fibrosis and, eventually, diabetic kidney disease itself [6–8].

This is why detecting DKD early matters so much — it buys time to slow disease progression and head off cardiovascular complications. Of the biomarkers available, the urinary albumin-to-creatinine ratio (UACR) has become the go-to screening test, being simple, cheap, non-invasive, reproducible, and able to

catch kidney injury before eGFR has meaningfully dropped. A persistently raised UACR reflects greater glomerular permeability and endothelial damage, making it one of the earliest signs of DKD that can actually be picked up clinically [9–12].

Both the ADA and KDIGO now call for UACR and eGFR to be checked together at the point T2DM is diagnosed, and then at least once a year afterward. Reading albuminuria and eGFR side by side sharpens risk stratification, opens the door to earlier treatment, and helps guide the choice of renoprotective agents — ACE inhibitors, ARBs, SGLT2 inhibitors, GLP-1 receptor agonists, and the non-steroidal mineralocorticoid receptor antagonists among them [13–16]. Starting these therapies early has been shown to meaningfully slow CKD progression, bring albuminuria down, and cut cardiovascular morbidity and mortality.

Studies from 2015 through 2026 keep landing on the same conclusion: a raised UACR at the point of T2DM diagnosis forecasts a faster decline in kidney function along with greater cardiovascular risk, more hospital admissions, and higher all-cause mortality. And when albuminuria comes down with treatment, renal outcomes tend to improve alongside it — underscoring why routine UACR screening matters so much in newly diagnosed patients [17–20].

This review brings together current evidence on what UACR contributes to early DKD detection in newly diagnosed T2DM. It also covers the pathophysiology behind albuminuria, present-day screening guidance, the key clinical studies from 2015 to 2026, recent therapeutic developments, and where renal outcomes in diabetes might be headed next [21].

2. Epidemiology of Diabetic Kidney Disease

DKD sits among the most common and most serious microvascular complications of diabetes, and it remains the world's leading cause of both CKD and ESKD. As T2DM becomes more common globally — pushed along by urbanisation, sedentary living, obesity, and an ageing population — the burden of DKD has grown right alongside it [1–3].

The IDF Diabetes Atlas puts the current global figure at over 589 million adults living with diabetes, a number set to climb considerably over coming decades. India carries one of the largest diabetic populations anywhere, which makes DKD a significant public health issue there specifically. Roughly 30 to 40% of T2DM patients go on to show some kidney involvement, though how common this is varies with age, ethnicity, how long someone has had diabetes, glycaemic control, hypertension, obesity, and genetic factors [1,2,4].

Several epidemiological studies point to DKD already being present at the point T2DM is diagnosed. Given that type 2 diabetes so often goes undiagnosed for years, prolonged hyperglycaemia can cause silent kidney damage well before anyone notices. Reported figures suggest 10–20% of newly diagnosed T2DM patients already have microalbuminuria at diagnosis, with a smaller group presenting with macroalbuminuria or a reduced eGFR [5–8].

How common albuminuria is climbs steadily the longer someone has had diabetes and the worse their metabolic control. Raised HbA1c, poorly controlled blood pressure, dyslipidaemia, smoking, obesity, and a family history of kidney disease have all held up as independent risk factors for DKD developing and worsening. And albuminuria isn't just a sign of kidney damage — on its own, it independently predicts cardiovascular morbidity and mortality too [9–12].

Multinational cohort data shows the DKD burden is still rising despite better diabetes care overall. Newer agents such as SGLT2 inhibitors and the non-steroidal mineralocorticoid receptor antagonists have helped renal outcomes, but late diagnosis remains a real obstacle, especially where routine screening is patchy, as is often the case in lower- and middle-income settings [13–16]. Indian hospital- and community-based studies report microalbuminuria rates in T2DM patients spanning 20% to 45%, the range reflecting differences in study population and diagnostic criteria. Poor glycaemic control, late diagnosis, limited healthcare access, and low awareness of kidney disease all add up to a substantial DKD burden across the Indian population specifically [17–20]. Catching this early through routine UACR testing offers a chance to identify patients while their kidney injury is still reversible. A persistently elevated UACR predicts progressive decline in kidney function, cardiovascular events, hospitalisation, and premature death — which is why international guidelines now call for UACR testing at T2DM diagnosis and at least yearly afterward, both to catch DKD early and to stratify risk [21–24].

Epidemiological evidence also indicates that pairing UACR with eGFR predicts renal and cardiovascular outcomes better than either measure alone. Patients whose eGFR is still normal but whose UACR is raised remain at meaningfully higher risk of CKD progression down the line — a reminder of how much value albuminuria carries as an early warning sign [25–28].

Large trials over the past decade have only reinforced how much prognostic weight UACR carries. When treatment brings albuminuria down, kidney function tends to decline more slowly, progression to ESKD is less likely, and cardiovascular mortality falls too — all of which speaks to the value of screening early in newly diagnosed T2DM [29].

3. Pathophysiology of Diabetic Kidney Disease and Mechanisms of Albuminuria

DKD arises from a mix of persistent hyperglycaemia, metabolic dysregulation, altered renal haemodynamics, chronic inflammation, oxidative stress, and several molecular pathways that gradually damage the glomeruli, tubules, interstitium, and vasculature of the kidney. Because these changes start years ahead of an actual T2DM diagnosis, it's not surprising that many newly diagnosed patients already show signs of kidney injury — albuminuria chief among them [6–9].

3.1 Persistent Hyperglycemia and Renal Injury

Chronic hyperglycaemia is what sets DKD in motion in the first place. Higher blood glucose ramps up intracellular glucose metabolism within renal cells, generating excess reactive oxygen species (ROS). This oxidative stress then damages glomerular endothelial cells, podocytes, mesangial cells, and tubular epithelial cells alike, leaving the kidney structurally and functionally impaired [6,10,11].

Ongoing hyperglycaemia also drives the formation of advanced glycation end products (AGEs), which build up in the glomerular basement membrane (GBM) and mesangial matrix. Once AGEs bind their receptors (RAGE), inflammatory cytokines and profibrotic mediators such as TGF-β get switched on, speeding up renal fibrosis and glomerulosclerosis [10–13].

3.2 Glomerular Hyperfiltration

Glomerular hyperfiltration is among the earliest changes seen in DKD. Because SGLT2 pulls more glucose back into the proximal tubules, less sodium reaches the macula densa, which — through altered tubuloglomerular feedback — dilates the afferent arteriole. Intraglomerular pressure then rises, putting mechanical strain on the filtration barrier and progressively injuring the glomerulus [12–15].

In the early stages, this hyperfiltration can actually keep eGFR looking normal or even higher than expected. Over time, though, sustained intraglomerular hypertension wears down the glomerular capillary wall, making it more permeable and letting albumin leak into the urine [13–16].

3.3 Podocyte Injury

Podocytes are specialised epithelial cells whose job is holding the glomerular filtration barrier together. Under the combined pressure of hyperglycaemia, oxidative stress, and inflammatory signalling, they undergo hypertrophy, apoptosis, and eventually detach from the basement membrane. Once enough podocytes are lost, the slit diaphragm breaks down, letting far more albumin through and producing albuminuria [14–18].

Podocyte loss is generally regarded as irreversible, and it tracks closely with both worsening renal function and advancing diabetic kidney disease [16–19].

3.4 Endothelial Dysfunction

Glomerular endothelial cells take damage early on too. Hyperglycaemia cuts nitric oxide production while pushing up oxidative stress, and the resulting endothelial dysfunction, along with damage to the endothelial glycocalyx, makes the vasculature leakier — letting albumin cross the glomerular capillary wall more easily [17–20].

Some recent evidence even suggests endothelial dysfunction shows up before albuminuria becomes clinically detectable, which strengthens the case for intervening early — before the structural damage becomes permanent [18–21].

3.5 Mesangial Expansion and Basement Membrane Thickening

These are classic histopathological findings in DKD, and they shrink the available filtration surface, steadily eroding renal function [11,19,22].

Further along, nodular glomerulosclerosis (the classic Kimmelstiel–Wilson lesions), tubular atrophy, interstitial fibrosis, and arteriolar hyalinosis all become more prominent [20–23].

3.6 Inflammation and Oxidative Stress

Low-grade chronic inflammation is now understood to be a major driver of DKD progression in its own right. Hyperglycaemia switches on NF-κB, which triggers release of inflammatory cytokines — TNF-α, IL-1β, and IL-6 among them — and these go on to promote endothelial injury, fibrosis, and tubular damage [21–25].

At the same time, the excess reactive oxygen species being generated amplifies inflammation, mitochondrial dysfunction, and apoptosis further still, speeding renal injury along [22–26].

3.7 Activation of the Renin–Angiotensin–Aldosterone System (RAAS)

RAAS activation sits at the centre of DKD's pathophysiology. Angiotensin II raises intraglomerular pressure, drives vasoconstriction, stimulates inflammatory cytokines, and, via TGF-β, accelerates fibrosis. Aldosterone adds to inflammation, oxidative stress, and extracellular matrix build-up on top of that [23–27].

The fact that ACE inhibitors and ARBs reliably lower albuminuria is itself good evidence for just how central RAAS activation is to DKD's pathogenesis [24–28].

3.8 Mechanism of Albuminuria

Albuminuria itself comes down to the glomerular filtration barrier losing its selectivity. Ordinarily, the glomerular endothelium, basement membrane, and podocyte slit diaphragm together keep albumin out of the urine almost entirely. Once diabetes has damaged

these structures, though, albumin filters through into the urinary space far more freely [25–30].

UACR captures this by measuring urinary albumin excretion while adjusting for how concentrated the urine sample is, using creatinine as the reference point. A sustained rise in UACR is therefore taken as one of the earliest clinical signals of DKD, often showing up years before eGFR starts to fall [26–32].

3.9 Clinical Significance of Albuminuria

Albuminuria's significance has moved well beyond just flagging kidney damage. A large body of research now shows that a raised UACR independently predicts progression to CKD, ESKD, cardiovascular events, stroke, heart failure, and all-cause mortality [30–36].

Notably, when treatment with RAAS inhibitors, tend to improve alongside it. Albuminuria has therefore ended up serving double duty — both as a diagnostic marker and as a treatment target in its own right for DKD patients [32–40].

4. Urinary Albumin-to-Creatinine Ratio (UACR): Principles, Measurement, Interpretation, and Clinical Utility

UACR has become the preferred screening tool for spotting DKD early, thanks to being simple, dependable, cheap, and capable of picking up kidney damage before eGFR has measurably dropped. Where 24-hour urine collection is cumbersome and error-prone, UACR needs only a single spot sample, correcting for urine concentration by expressing albumin as a ratio to creatinine [9–12].

4.1 Principle of UACR

Under normal conditions, only a trace of albumin gets through the glomerular filtration barrier at all, thanks to an intact basement membrane, endothelial glycocalyx, and podocyte slit diaphragm — and whatever does get filtered is almost entirely reabsorbed by the proximal tubule, leaving urinary albumin excretion very low [10,13].

In diabetes, though, persistent hyperglycaemia, glomerular hypertension, oxidative stress, and inflammation all chip away at this barrier, so more albumin leaks into the urine. Because a raw urinary albumin measurement can be thrown off by hydration and how dilute the sample is, albumin is instead expressed against urinary creatinine — giving a standardised, accurate read on albumin excretion regardless of dilution [11–15].

4.2 Sample Collection

For routine screening, an early-morning first-void midstream sample is the preferred choice, since it minimises the influence of posture, activity, and hydration. That said, a random spot sample is considered perfectly acceptable in everyday clinical practice, and both ADA and KDIGO guidelines endorse it [13–16].

Transient elevations in urinary albumin may occur due to:

- Urinary tract infection
- Fever
- Vigorous exercise
- Menstruation
- Severe hypertension
- Acute illness
- Heart failure
- Pregnancy

Given this, an abnormal UACR result calls for confirmation — repeating the test within 3–6 months, with at least two of three samples staying abnormal before persistent albuminuria is actually diagnosed [14–18].

4.3 Laboratory Methods for UACR Estimation

Urinary albumin can be measured using several analytical techniques, including:

- Immunoturbidimetric assay
- Immunonephelometric assay
- Immunoassay-based automated analyzers
- High-performance liquid chromatography (HPLC)
- Liquid chromatography–mass spectrometry (mainly for research)

Creatinine itself is usually measured via the Jaffe reaction or enzymatic assays, and automated immunoturbidimetric methods dominate routine clinical labs because they're accurate, reproducible, quick, and inexpensive to run [15–19].

4.4 Interpretation of UACR

KDIGO and ADA guidelines split albuminuria into three categories, as follows:

Category	UACR (mg/g creatinine)	Clinical Interpretation
A1	<30	Normal to mildly increased
A2	30–300	Moderately increased albuminuria (formerly microalbuminuria)
A3	>300	Severely increased albuminuria (formerly macroalbuminuria)

When albuminuria in the A2 or A3 range persists beyond three months, that's taken as evidence of chronic kidney disease [16–20].

4.5 Clinical Utility of UACR

In newly diagnosed T2DM, UACR earns its place through several distinct clinical uses:

1. Early Detection of Diabetic Kidney Disease

Albuminuria frequently shows up years ahead of any drop in eGFR, so UACR can flag early renal injury while kidney function still looks entirely normal — leaving room for intervention while it still matters most [17–21].

2. Risk Stratification

Higher UACR levels correlate strongly with:

- Progressive decline in eGFR
- Chronic kidney disease progression
- End-stage kidney disease
- Cardiovascular disease
- Heart failure
- Stroke
- All-cause mortality

Even a modest rise in albuminuria has been tied to worse cardiovascular outcomes [18–23].

3. Monitoring Disease Progression

Tracking UACR over time helps clinicians judge how the disease is progressing and how well treatment is working — a fall in albuminuria after starting therapy tends to go hand in hand with a better renal prognosis and lower cardiovascular risk [20–25].

4. Guiding Therapeutic Decisions

Identification of persistent albuminuria supports initiation or optimization of renoprotective therapies, including:

- ACE inhibitors
- Angiotensin receptor blockers (ARBs)
- Sodium-glucose cotransporter-2 (SGLT2) inhibitors
- Glucagon-like peptide-1 receptor agonists (GLP-1 RAs)
- Non-steroidal mineralocorticoid receptor antagonists (e.g., finerenone)

Together, these agents cut albuminuria, slow CKD progression, and improve cardiovascular outcomes [22–27].

4.6 Advantages of UACR

The major advantages of UACR include:

- Simple and non-invasive test
- Requires only a spot urine sample
- No need for 24-hour urine collection
- Cost-effective and suitable for large-scale screening
- Highly sensitive for detecting early diabetic kidney disease
- Easily reproducible
- Recommended by international guidelines
- Useful for monitoring treatment response [23–30].

4.7 Limitations of UACR

Despite its clinical utility, UACR has several limitations:

- Biological variability between samples
- Temporary elevation during acute illness or exercise
- False-positive results in urinary tract infection or menstruation
- Albuminuria may be absent in non-albuminuric diabetic kidney disease
- Requires confirmation with repeat testing
- Should always be interpreted together with eGFR and clinical findings [26–34].

4.8 Current Guideline Recommendations

The American Diabetes Association (ADA) and KDIGO recommend:

- Measuring UACR and eGFR at the time of diagnosis in all patients with T2DM.
- Repeating both tests at least annually.
- Confirming abnormal UACR with repeat testing over 3–6 months.
- Using both UACR and eGFR for CKD staging and risk stratification.
- Initiating kidney-protective therapy in patients with persistent albuminuria and appropriate clinical indications [31–40].

Taken together, UACR still stands as the gold-standard first screening test for early DKD, given how simple and accessible it is, how much prognostic weight it carries, and its ability to flag renal injury before kidney function is lost for good.

MATERIALS AND METHODS

Search terms combined MeSH headings with keywords such as “Type 2 Diabetes Mellitus,” “Diabetic Kidney Disease,” “Diabetic Nephropathy,” “Urinary Albumin-to-Creatinine Ratio,” “UACR,” “Albuminuria,” “Microalbuminuria,” “Chronic Kidney Disease,” “Early Screening,” “Kidney Biomarkers,” and “Renal Outcomes,” linked with Boolean AND/OR operators to sharpen the results.

Excluded were paediatric studies, animal experiments, case reports, conference abstracts without full text, duplicate publications, and non-English articles. Titles and abstracts were screened first, with full texts pulled for anything that passed that initial filter. Data extraction focused on study characteristics, patient population, how common albuminuria was, UACR's diagnostic accuracy, its prognostic weight, its links to renal and cardiovascular outcomes, and current screening guidance — all synthesised narratively.

Forty key references from 2015 to 2026 were ultimately chosen, selected for methodological quality, clinical relevance, and how much they added to understanding UACR as an early biomarker of DKD in newly diagnosed T2DM.

RESULTS

The literature search turned up studies assessing UACR's role in early DKD detection among T2DM patients. After eligibility screening, 40 publications from 2015–2026 made it into this review Most of the studies included agreed that UACR is a sensitive, dependable marker for catching DKD early, often flagging renal involvement well before eGFR meaningfully drops [5–12]. A number of them reported that 10–30% of newly diagnosed T2DM patients already had moderately increased

albuminuria (UACR 30–300 mg/g) — evidence that kidney damage can already be present right at the point of diabetes diagnosis [8–14].

Most observational studies linked a raised UACR to poor glycaemic control specifically — higher HbA1c, longer periods of undiagnosed hyperglycaemia, hypertension, obesity, dyslipidaemia, and smoking all came up repeatedly [10–18]. Higher UACR values also tracked with a faster fall in renal function, greater risk of CKD progression, and more cardiovascular events and all-cause deaths [15–26].

Trials of renoprotective therapies — ACE inhibitors, ARBs, SGLT2 inhibitors, GLP-1 receptor agonists, and finerenone among them — found meaningful reductions in albuminuria and slower DKD progression, reinforcing just how much early UACR screening matters clinically [20–35].

The most recent ADA and KDIGO guidelines both continue to recommend UACR alongside eGFR at T2DM diagnosis and at least once yearly afterward, for early detection and risk stratification of DKD [36–40].

Altogether, this evidence makes a strong case for UACR as the default initial screening biomarker for DKD in newly diagnosed T2DM, given how simple, non-invasive, affordable, and prognostically useful it has proven to be.

DISCUSSION

DKD is still one of the more serious microvascular complications tied to T2DM, and it remains a leading global cause of CKD and ESKD [1,2]. What this review shows is that UACR holds up as a simple, sensitive, clinically useful biomarker for catching renal involvement early in newly diagnosed T2DM. Evidence spanning 2015 to 2026 keeps backing the routine use of UACR to spot kidney damage before eGFR has meaningfully declined [3–6].

One of the more striking things to come out of this literature is just how many patients already have albuminuria at the point T2DM is diagnosed. Since hyperglycaemia can sit unnoticed for years, structural and functional kidney changes have plenty of time to develop before diabetes is ever formally recognised. Several of the included studies put the figure at 10–30% of newly diagnosed patients already showing moderately increased albuminuria (UACR 30–300 mg/g), which argues for screening right at the point of diagnosis rather than waiting [7–10]. There's also a clear link between raised UACR and poor glycaemic control — higher HbA1c consistently tracked with greater urinary albumin excretion, fitting with the idea that chronic hyperglycaemia drives glomerular endothelial dysfunction, oxidative stress, podocyte injury, and inflammatory activation, all of which end in albuminuria [11–15]. That reinforces just how much early, sustained glycaemic control matters for keeping DKD risk down. Hypertension came up repeatedly too as a major driver of raised UACR — higher systemic and intraglomerular pressure both speed glomerular injury and push albuminuria along, which is why tight blood pressure control through RAAS inhibitors remains a cornerstone of renoprotective care in albuminuric diabetic patients [16–20].

Beyond diagnosis, UACR has also proven itself a strong prognostic marker. A number of cohort studies found that rising UACR independently predicts eGFR decline, CKD progression, ESKD, cardiovascular events, heart failure, and all-cause death [21–26]. Even patients whose eGFR is still preserved carry substantially higher risk if their UACR is elevated, which is exactly why UACR and eGFR work best used together for risk stratification.

Newer therapies have only added to UACR's clinical relevance. Large randomized trials of SGLT2 inhibitors, GLP-1 receptor agonists, and the mineralocorticoid receptor antagonist finerenone all show meaningful reductions in albuminuria and slower CKD progression in T2DM [27–33]. And because albuminuria responds to treatment, tracking UACR serially is useful well beyond diagnosis — it also helps monitor how well therapy is working and how the disease is progressing.

Both ADA and KDIGO guidelines currently call for UACR and eGFR together at T2DM diagnosis, repeated at least yearly [34–38]. The evidence gathered here backs those recommendations solidly, and points to routine UACR testing becoming a standard part of diabetes care generally — especially in resource-limited settings, where catching disease early can meaningfully cut the burden of advanced kidney disease down the line. UACR isn't without its limits, though. It can rise transiently with a urinary tract infection, fever, hard exercise, dehydration, menstruation, or acute illness, so persistent albuminuria always needs confirming with repeat testing before a DKD diagnosis is actually made [39]. And some patients go on to develop non-albuminuric DKD, where kidney function declines without albuminuria at all — a reminder that UACR should never be read in isolation from eGFR and the wider clinical picture [40].

All told, this evidence confirms UACR as about the most practical, clinically useful biomarker available for catching DKD early in newly diagnosed T2DM. Screening for it routinely opens the door to timely intervention, sharper risk stratification, better-guided renoprotective therapy, and, potentially, improved long-term renal and cardiovascular outcomes.

Literature from 2015 through 2026 consistently places albuminuria among the earliest detectable signs of diabetic renal involvement, often well ahead

of any measurable eGFR decline [3–6] — which lines up neatly with ADA and KDIGO's continued recommendation to screen with both UACR and eGFR at T2DM diagnosis and yearly thereafter [7,8].

A number of the epidemiological studies here reported roughly 10–30% of newly diagnosed T2DM patients already presenting with moderately increased albuminuria [9–12]. This fits with how long type 2 diabetes can stay asymptomatic, during which time chronic hyperglycaemia is quietly damaging the renal microvasculature well before the disease gets recognised clinically. Waiting for symptoms before assessing renal function risks missing the window where early intervention would do the most good.

Sustained hyperglycaemia stays the chief pathogenic driver behind DKD. Both experimental and clinical work show that chronically high blood glucose fuels oxidative stress, AGE formation, endothelial dysfunction, podocyte injury, inflammation, and RAAS activation — all of which raise glomerular permeability and drive albuminuria [13–17]. A raised UACR, then, reflects early glomerular injury and doubles as a useful marker of ongoing kidney damage.

There's also a solid relationship in this evidence between raised UACR and poor glycaemic control specifically. Multiple observational studies found urinary albumin excretion correlating positively with HbA1c, suggesting that poor glycaemic control speeds up diabetic nephropathy [18–21]. Hypertension, obesity, dyslipidaemia, smoking, and older age all came up repeatedly too, as risk factors tied to higher UACR and a faster drop in renal function [22–25].

One notable finding from recent literature is that albuminuria isn't only a kidney marker — it's a strong cardiovascular predictor too. Several cohort studies link rising UACR independently to myocardial infarction, stroke, heart failure, cardiovascular death, and all-cause mortality, even in patients whose kidney function is otherwise intact [26–29]. Routine UACR testing, in other words, gives prognostic information that extends well beyond the kidneys alone.

Recent therapeutic developments have underscored just how much early albuminuria detection matters clinically. Large randomized trials of SGLT2 inhibitors, GLP-1 receptor agonists, and finerenone all show meaningful reductions in albuminuria and slower CKD progression in T2DM patients [30–34] — evidence that catching a raised UACR early lets clinicians start renoprotective therapy sooner, with knock-on benefits for both renal and cardiovascular outcomes long-term.

For all its strengths, UACR has real limitations. Results vary biologically between samples and can spike transiently with a urinary infection, fever, strenuous exercise, menstruation, dehydration, or acute illness [35], which is why guidelines call for repeat testing over 3 to 6 months before persistent albuminuria is confirmed [36]. There's also a growing recognition that some diabetic patients develop non-albuminuric kidney disease, where renal function worsens despite a normal UACR [37,38] — so UACR always needs to be read alongside eGFR and the broader clinical picture, never on its own.

On the whole, this evidence backs UACR firmly as the preferred first screening biomarker for DKD in newly diagnosed T2DM. Its simplicity, accessibility, diagnostic accuracy, and prognostic weight together make it hard to leave out of routine diabetes care. Paired with eGFR and sound clinical judgement, it lets clinicians identify at-risk patients early, start evidence-based therapy promptly, and track disease progression effectively — ultimately lightening the load of both chronic kidney disease and cardiovascular complications [40].

CONCLUSION

DKD is a major microvascular complication of T2DM, and it very often gets underway before diabetes is even formally diagnosed. The evidence surveyed here, spanning 2015 to 2026, shows UACR to be a simple, sensitive, non-invasive, cost-effective biomarker for catching renal involvement early in newly diagnosed T2DM patients. Checking UACR routinely, alongside eGFR, lets clinicians identify patients at risk of CKD progression and cardiovascular complications early enough to start renoprotective therapy in good time. Both the evidence and international guidelines firmly back UACR as the preferred screening tool for DKD, both at T2DM diagnosis and through ongoing follow-up, with the aim of improving long-term renal and cardiovascular outcomes [1–40].

Limitations

- As with any narrative review, the conclusions here are only as strong as the quality and consistency of the underlying published studies.
- Differences in study populations, sample sizes, follow-up length, and how UACR was measured across studies may limit how directly their outcomes can be compared.
- And while evidence through 2026 is included here, newer studies and future guideline updates will likely refine UACR's role in diagnosing and managing DKD further still.

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