



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Review Article

Review on Diabetic Kidney Disease

Yuvraj Rattu, Rajesh Kumar, Ajeet Pal Singh, Amar Pal Singh, Gaurav Hastir

St. Soldier Institute of Pharmacy, Lidhran Campus Behind NIT (R.E.C) Jalandhar- Amritsar byepass, NH-1, Jalandhar, Punjab, India 144011

ARTICLE INFO

Published: 11 Sept 2026

Keywords:

Diabetic Kidney Disease (DKD), Diabetes Mellitus, Kidney Damage, Early Diagnosis, Biomarkers for Disease Progression.

DOI:

10.5281/zenodo.22704747

ABSTRACT

Diabetic Kidney disease or DKD is a common condition that worldwide affects a large number of Diabetics. Diabetic kidney disease arises when diabetes-related blood sugar level raises and simultaneously harms the Kidneys. Harmful consequences leads to change in blood flow and pressure inside the kidneys and cause inflammation throughout the body due to excessive Sugar and fat metabolism in blood. An obstacle faced during the accurate diagnoses of DKD is that it shows the same symptoms as other kidney disorders. So in order to accurately diagnose DKD, Physician requires newly improved testing that can monitor its progression, determine whether therapies are effective and predict potential future outcomes. Initial cause is blood sugar level, which cause the body to generate an excessive amount of harmful molecules when blood sugar level is high and function inside cells and tissues like a wrecking ball. Most of the people are unaware of its potential danger. It causes and a large of people are particularly unaware of even suffering from DKD, despite the fact that it is a major health concern. Another significant issue of DKD is its High expense treatment which is a major concern for patients, their families, healthcare system and economies. In the early stages most people feel completely normal until the kidney damage is extremely severe there are typically no noticeable symptoms and by then major consequences have frequently begun. Another important worry is the lack of good statistics on the incidence of diabetic kidney disease in many places, as well as the absence of systematic screening for kidney abnormalities in diabetes patients, which frequently leads to the disease being found too late. Doctors could delay or stop the damage to help individuals. It's early diagnose will help the patient live a more longer and healthier lives as well as reducing the financial burden and emotional impact on patients and their families. Early diagnose will help physicians to provide a more appropriate medication and environment condition in which they can recover faster. But unfortunately DKD is often identified when kidney are for beyond damage and close to failure.

***Corresponding Author:** Yuvraj Rattu

Address: St. Soldier Institute of Pharmacy, Lidhran Campus Behind NIT (R.E.C), NH-1, Jalandhar, Punjab, India 144011

Email ✉: yuvrajrattu407@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



INTRODUCTION

Diabetic kidney disease is a kidney condition that is caused by diabetes which is going to be discussed in this article. A major factor which aggravate this illness is inflammation or swelling and irritation within the kidney [1]. Early detection of bodily inflammatory markers are crucial. We can prevent the issue from worsening only by promptly identifying the warning indicators or symptoms before it causes any significant harm [2].

The Diabetic kidney disease is also known as diabetic nephropathy. It is a unique kind of kidney disease that only affects individuals with diabetes who also experience chronic kidney damage [3]. changing how the kidneys work as well as how the body eliminates waste and fluids. Albuminuria, edema, polyuria, nausea, anemia, and hypertension are the signs of this illness [4].

For those peoples who already have diabetes, this kidney issue raises their risk of dying by about 31%. The risk increases with the severity of kidney disease [5]. Early detection of diabetic kidney disease (DKD) is crucial since it not only saves a significant amount of money for families and the nation as a whole, but it also lessens the considerable harm that DKD does to people's health. Early detection allows medical professionals to begin therapies that slow down kidney deterioration and avoid more serious issues down the road. DKD is frequently concealed and initially undetectable; most people experience no symptoms at all for a considerable amount of time.

Early detection of diabetic kidney disease, or DKD, is a wise and economical strategy to lessen the total damage and consequences of the condition. This study examines the prevalence of DKD worldwide, the primary risk factors that raise the likelihood of developing it, and potential early

warning indicators that manifest before the condition worsens [6].

Epidemiology and Perspectives in DKD

At a global level, diabetes is a relatively concerning health issue. At present at least 10.5% of people are suffering from diabetes, mostly aged between 20 years to 75 years. According to experts by 2030 this figure will significantly increase and cross over 643 million [7]. Diabetes usually goes undiagnosed for the first four to seven years and by the time physician diagnose it a significant damage has already been done to the body which remains undetected as its symptoms doesn't manifest until much later [8]

This illness worsens over time due to a number of internal factors, including persistent inflammation, irregularities in the body's handling of chemicals and energy (metabolic disorders), and problems with blood flow and pressure (hemodynamic issues). Since focusing on and managing the inflammatory process appears to be one of the most promising approaches to create novel treatments and halt the progression of the disease, scientists and medical professionals have been paying much more attention to this aspect lately [9].

Diabetic kidney disease, affects around 38.8% of people with diabetes in China. However, only about 2.9% of the overall population—including those with diabetes—has this illness in rural areas [10]. DKD affected 34.4% of Indians and 62.3% of participants in a multicenter trial [11]. Diabetes and chronic renal disease are most prevalent among older adults in the Eastern Mediterranean region. About 15.3% of type 2 diabetes patients in Japan have a low estimated glomerular filtration rate (eGFR), which indicates that their kidneys are damaged or functioning less well [12]. Approximately 28% of individuals with type 2



diabetes in the UK experienced renal issues after being followed for approximately 15 years, according to a significant long-term study. Diabetic kidney disease, or kidney impairment brought on by diabetes, affects around 2.2% of the US population overall. As more individuals get diabetes, this percentage has been rising [13]. The study highlights how critical it is to increase the public awareness for diabetic kidney disease and increase efforts to prevent it.

Diabetic kidney disease is a highly common and dangerous problem in the world. Approximately 1.75 out of every 100 patients with diabetes (or roughly 1.75%) acquire this illness, which causes kidney damage over time [14]. Different rates of DKD have been found in China, India, and the United Arab Emirates, according to research.

Pathophysiology of DKD

Inflammatory, haemodynamic, and metabolic factors interact in a complex way to cause diabetic kidney disease [15]. Endothelial dysfunction, oxidative stress, tubule and glomerulus damage, and other metabolic responses are triggered by hyperglycemia [16]. While renal hyperfiltration and increased intraglomerular pressure cause glomerular injury, tubular dysfunction impacts the reabsorption and secretion processes [17]. Chronic inflammation and fibrosis exacerbate renal damage and cause a gradual decline in kidney function [12]. In order to preserve the renal function and improve diabetic kidney disease outcomes, as it is intended by developing therapies which require a considerable amount of understanding about these pathophysiological processes.

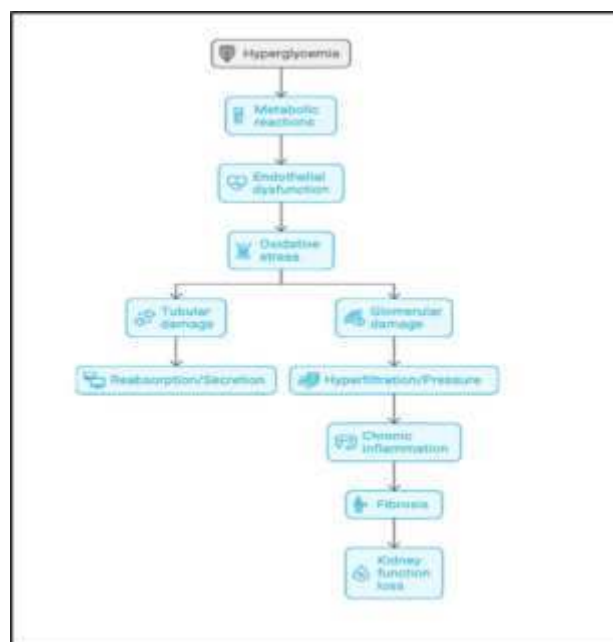


Figure 1: 1,2 The Pathophysiology of Diabetic Kidney

A detailed information regarding the various factors that lead to the beginning and progression of diabetic kidney disease, further highlighting the concerning issue and need for the comprehensive care techniques.

1. **Dyslipidemia:-** Endothelial dysfunction and kidney injury is carried by abnormal lipid metabolism
2. **Hypertension:-** Due to the high blood pressure the blood vessels in the kidneys got damaged.
3. **Hyperglycemia:-** Kidneys are largely damaged with the high blood sugar level which further alter metabolism as well.
4. **Oxidative stress:-** It is a phenomenon which is caused due to imbalance between the reactive oxygen species and the antioxidant.
5. **Genetic Predisposition:-** Diabetics are generally prone to genetically inherit kidney illness.

6. Inflammation:- Continuous and persistent low grade Inflammation further aggravates renal impairment.
7. Protein Kinase C Activation:- By dysfunction of protein kinase C signalling pathways, damage is caused to the kidneys.
8. Renin-Angiotensin System Activation:- An overactive renin-angiotensin system can bring vasoconstriction and fibrosis in the kidneys.
9. Advanced Glycation End Products (AGEs):- Advanced glycation end products get accumulated which causes Inflammation and endothelial impairment.
10. Endothelial dysfunction:- Vascular diseases are resulted in due to endothelial dysfunction.
11. Podocyte Injury:- Podocyte destruction is caused due to the degradation of glomerular filtration barrier.
12. Renal Hyperfiltration:- High damage is done to the glomerular which can be caused in the elevation of glomerular filtration rate.
13. Tubulointerstitial Fibrosis:- Renal fibrosis is resulted in due to the excessive accumulation of extracellular matrix proteins.
14. Autoimmunity:- In vulnerable people immune mediation can cause damage to kidney tissue.
15. Environmental Factors:- the progression of this disease can be caused by the dirt, lifestyle and other environmental factors [18].

Polyol, hexosamine, advanced glycation end products and protein kinase C are made to use in metabolic pathways [19]. Kidney damage is caused by hyperglycemia which produces

glycosylated end products, synthesising diacylglycerol and activating protein C [20].

Limitations of Conventional Biomarkers

Diagnostic indicators of diabetic kidney disease are albuminuria and a significant decline in estimated glomerular filtration rate (eGFR) as it is measured by blood creatinine levels [21]. To predict future progression and assess treatment success is done by using albuminuria as a valid biomarker. An important tool in diagnosing and treating diabetic kidney is urinary albumin excretion (UAE), that stratifies and to assess the severity of albuminuria [22].

A key clinical biomarker that is used to assess the prognosis of diabetic kidney disease is glomerular filtration rate (GFR). There is a clear link between structural damage and kidney function, even though this relationship is less obvious with modest albuminuria or a little change or decrease in glomerular filtration rate [23].

Changes in current and earlier estimated GFRs (eGFRs) are crucial markers of how the condition is expected to progress. The CKD-EPI and MDRD equations are frequently used to estimate glomerular filtration rates (GFR) [24]. It is crucial to note, however, that these formulae may underestimate or overestimate the actual GFR. This mismatch could be due to compensatory changes in the functioning of the remaining nephrons [25]. The diagnostic and follow-up approaches that are now in use have limitations, especially for people with the common normoalbuminuric phenotype who lack specific therapeutic alternatives [26].

The study emphasises the need for innovative biomarkers to improve diagnostic and monitoring procedures for diabetic kidney disease, with a focus on their efficacy in diagnosing, tracking



progression, assessing therapy response, and predicting prognosis [27]. The study will look into

the practical application of novel biomarkers as shown in Figure 2.

Sr. no	Conventional Biomarkers	Type	Description	Clinical Relevance
1	Albuminuria	Urinary	Presence of albumin in urine, indicative of kidney damage	Gold standard for DKD diagnosis and monitoring
2	Estimated Glomerular Filtration Rate (eGFR)	Blood/Calculation	Calculation of kidney function based on serum creatinine levels using equations such as MDRD or CKD-EPI	Assesses the degree of kidney function and stages of CKD
3	Neutrophil Gelatinase-Associated Lipocalin (NGAL)	Urinary	Marker of tubular injury and inflammation	Early detection of AKI and DKD progression
4	Kidney Injury Molecule-1 (KIM-1)	Urinary	Tubular injury marker associated with inflammation and fibrosis	Prognostic marker for DKD progression and renal function decline
5	Serum Cystatin C	Blood	Protein used to estimate GFR, independent of muscle mass	More accurate in detecting early declines in kidney function than creatinine
6	Urinary Biomarkers (e.g., MCP-1, TGF- β 1, IL-6)	Urinary	Inflammatory and fibrotic markers reflecting renal damage	Potential for early detection and risk stratification
7	High-Sensitivity C-Reactive Protein (hs-CRP)	Blood	Marker of systemic inflammation associated with DKD	Predictive of cardiovascular risk in DKD patients
8	FGF-23	Blood	Phosphate-regulating hormone associated with mineral metabolism and cardiovascular disease	Predictor of CKD progression and adverse outcomes in DKD
9	Urinary Exosomal microRNAs	Urinary	Small non-coding RNAs implicated in gene expression regulation	Potential for early detection and therapeutic monitoring
10	Urinary Albumin Excretion	Urinary	Measurement of albumin-to-creatinine ratio (UACR) in a spot urine sample	Early markers of kidney damage. Persistent albuminuria indicates DKD.
11	Serum Creatinine	Blood	Measurement of creatinine levels in the blood to estimate GFR	Elevated levels indicate reduced kidney function.
12	Blood Urea Nitrogen (BUN)	Blood	Measurement of nitrogen in the blood that comes from the waste product urea.	Elevated levels indicate impaired kidney function.
13	Urinary Total Protein	Urinary	Measurement of total protein in the urine.	Indicates the presence and extent of proteinuria.
14	Hemoglobin A1c (HbA1c)	Blood	Measurement of average blood glucose levels over the past 2-3 months.	Indicates glycemic control, a crucial factor in managing DKD.

Sr. no	Novel Biomarkers	Type	Description	Clinical Relevance
1	Urinary Retinol-Binding Protein (RBP)	Urinary	Marker of tubular proteinuria.	Indicates proximal tubular damage and early renal dysfunction.
2	Kidney Injury Molecule-1 (KIM-1)	Urinary	Tubular injury marker associated with inflammation and fibrosis	Prognostic marker for DKD progression and renal function decline
3	Serum soluble uricase/plasminogen activator receptor (sPAR)	Blood	Marker of systemic inflammation and immune activation	Associated with DKD development and progression
4	Urinary Liver-Type Fatty Acid-Binding Protein (L-FABP)	Urinary	Marker of proximal tubular injury and oxidative stress	Predictive of early DKD progression and adverse outcomes
5	Urinary Netrin-1	Urinary	Axonal guidance cue protein involved in tubular integrity and repair	Elevated levels associated with DKD progression and renal fibrosis
6	Plasma Soluble Endothelial Protein C Receptor (sEPCR)	Blood	Endothelial cell marker reflecting endothelial dysfunction	Predictive of renal and cardiovascular outcomes in DKD
7	Urinary Aquaporin-2 (AQP2)	Urinary	Water channel protein reflecting tubular function and urine concentration	Potential marker for DKD progression and response to therapy
8	Plasma Angiotensin-like 4 (ANGPTL4)	Blood	Lipid metabolism regulator associated with inflammation and endothelial dysfunction	Elevated levels predictive of DKD progression and cardiovascular events
9	Plasma Pentraxin-3 (PTX3)	Blood	Acute phase protein involved in innate immunity and inflammation	Associated with DKD severity and cardiovascular risk
10	Plasma Sema3	Blood	Hormone implicated in pancreatic beta-cell proliferation and insulin resistance	Elevated levels correlate with DKD severity and decline in renal function
11	Transforming Growth Factor-beta 1 (TGF- β 1)	Urinary/Blood	Marker associated with renal fibrosis and inflammation	Correlates with the severity of renal damage and progression of DKD.
12	N-Acetyl-D-Glucosaminidase (NAG)	Urinary	Enzyme marker of tubular damage.	Elevated levels indicate early tubular injury.
13	Plasma Beta-2 Microglobulin	Blood	Marker of tubular reabsorption efficiency.	Elevated levels are associated with tubular dysfunction and early kidney damage.
14	Urinary Angiotensinogen	Urinary	Marker associated with intrarenal RAAS activation.	Indicates early intrarenal angiotensin II activity and kidney injury.

Figure 2: 1, 2. This table presents a complete overview of conventional biomarkers [28], [29] versus innovative biomarkers [30], [31], [32] that are routinely used in clinical practice to diagnose, monitor, and manage diabetic kidney disease, with an emphasis on kidney function, damage, and overall metabolic management.

Novel Biomarkers in Diabetic Kidney Disease

In diabetes related disease, early detection is crucial but traditional diagnostic procedures led to frequent delay in diagnosis [33]. The study of DKD pathophysiology and causes has highlighted the necessity of developing new biomarkers in urine and serum using proteomics and metabolomics.

Biomarkers have been created that can help in the diagnosis of diabetic kidney disease, leading to further advancement. Serum creatinine, opposed

to the traditional ones, can improve the accuracy of DKD examinations, which makes them more of a precise and accurate instrument for monitoring the functions of the kidney [34].

The AKI agreement emphasises that biomarkers should serve as an extra test to identify the individuals who can benefit from the cardiovascular risk prevention and treatment techniques rather than replace it with the traditional testing and clinical evaluations altogether [35].



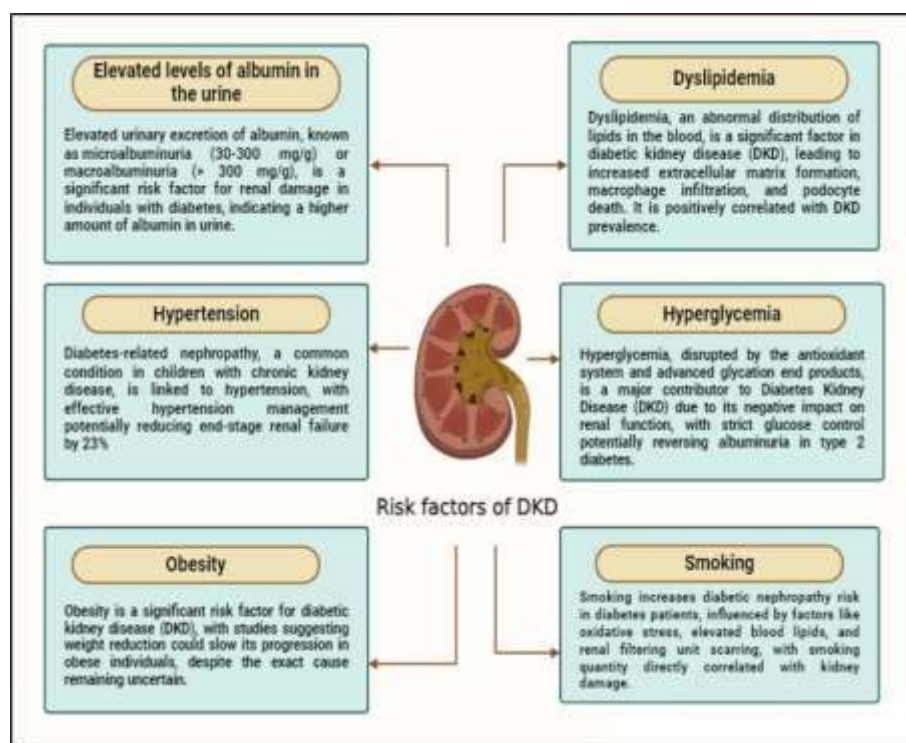


Figure 3: 1,2 Potential Risk factors of DKD [36], [37]

Biomarkers for the early diagnosis of DKD

To identify renal impairment prior to the onset of clinical signs, biomarkers are being used such as urine albumin excretion rate, UAER, UACR and eGFR in the diagnosis of diabetic kidney disease [6]. L-FABP, NGAL, and KIM-1 are examples of novel biomarkers that may be used to detect kidney damage early. To slow down the development of diabetic kidney disease and its consequences, early treatment and special tailored approaches are being employed including various indicators into clinical practice.

To properly treat kidney disease, timely diagnosis and appropriate medication are critical. They can

significantly reduce or slow down the progression of the illness as well as increase life expectancy and lessen the stress both mentally and physically, further relieving their financial stress [38]. Albuminuria and serum creatinine serve as biomarkers but their absence can hinder the timely diagnosis of a condition [39]. Although some research shows that albuminuria is an insensitive biomarker and unreliable even serum creatinine is not considered such a strong predictor [40]. As a result, new biomarkers with higher sensitivity and predictability are required for early diagnosis and treatment of DKD, as present biomarkers are insufficiently sensitive.

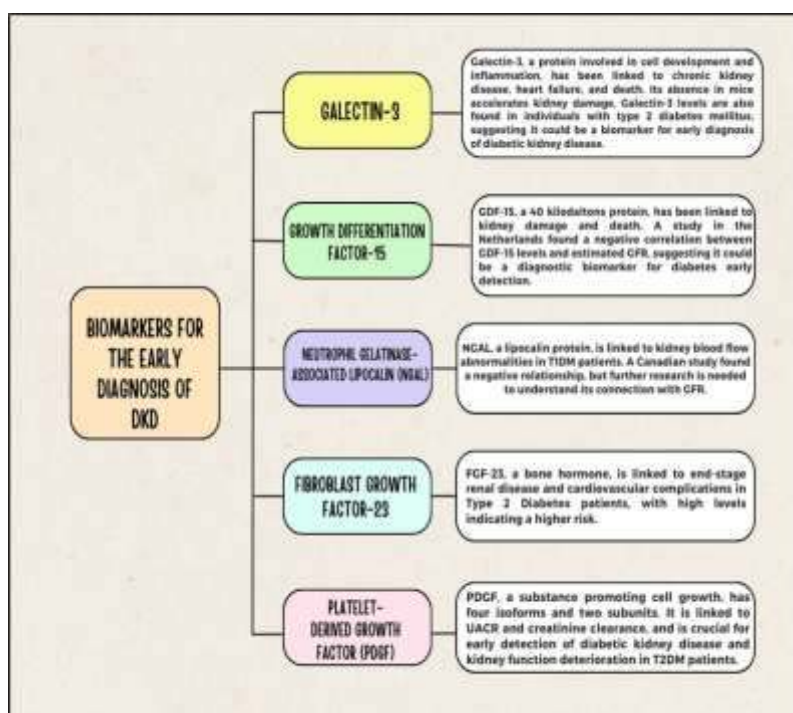


Figure 4: 1,2 Potential Biomarkers for the early diagnosis of DKD [41], [42], [43]

Diagnosis of DKD

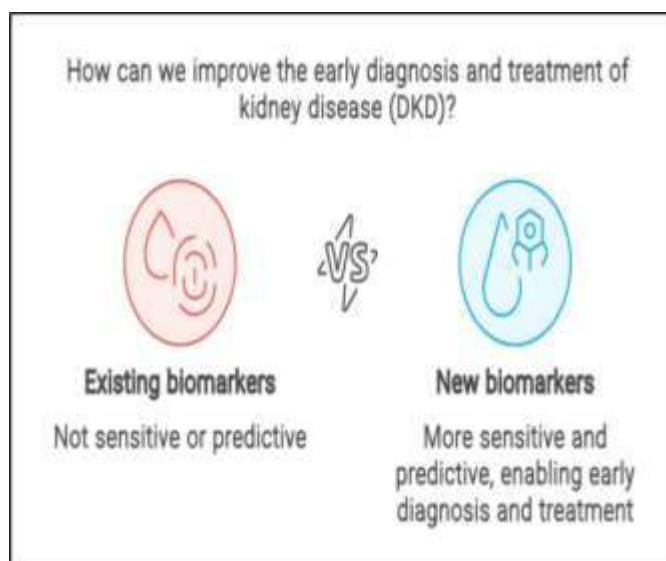


Figure 5: Type of biomarker are more important for disease diagnosis and treatment

Diabetic kidney disease is diagnosed by evaluating urine albumin excretion and glomerular filtration rate (GFR) [44]. While GFR monitoring and chronic albuminuria help to stage the seriousness of diabetic kidney disease, the early renal impairment is identified by albuminuria screening [45]. Further diagnostic techniques include kidney

biopsies, serum Biomarkers and renal imaging. Early detection and surveillance are essential for the prompt intervention and therapy to prevent diabetic kidney disease from further processing to end-stage renal disease.

Diabetic kidney disease is diagnosed using a variety of diagnostic procedures that aid in the

early discovery, in monitoring illness progress and directing decisions for the treatment. These diagnostic procedures specially emphasize the indicator of kidney damage, function and generally metabolic control.

Various diagnostic assessments used in the diagnosis of DKD

A. Screening

1. Annual Screening:- Those patients who are suffering from diabetes need to have a screening test of kidney disease.
2. Glomerular Filtration Rate:- To find out the estimated GFR various formulas are used such as CKD-EPI or MDRD. Indication of renal damage is an eGFR of less than 60ml/min/1.73m².
3. Urinary albumin excretion:- Ascot urine sample is used to determine the urinary albumin to creatine ratio (UACR). Renal injury is suggested by persistently in albuminuria (>30mg/g).

B. Diagnostic Confirmation

1. Measurement of Serum Creatinine: To determine GFR and evaluate renal function serum creatinine levels are measured.
2. Conformation of albumin: A minimum of two of every three urine samples are obtained over a time period of three to six months is used for confirmation testing.

C. Additional Tests

1. Renal Biopsy: In some particular situations, to confirm diagnoses and direction of treatment choices requires necessary renal biopsies.

2. Renal Imaging: In order to evaluate kidney anatomy and spot anatomical anomalies requires imaging test including ultrasonography.
3. Serum Biomarkers: Urine biomarkers such as NGAL, KIM-1 or serum cystatin C is measured to learn more about kidney damage and its prognosis.

D. Evaluation of Complications

1. Screening for Other Complications: Regular screening is needed for disease related problems such as retinopathy and neuropathy for any further complications.
2. Cardiovascular Assessment:- Considering the significant risk of cardiovascular illness; constant evaluation of cardiovascular risk factors is needed in DKD, such as hypertension and dyslipidaemia.

E. Monitoring and Follow-up

1. Multidisciplinary Care: To provide comprehensive therapy of diabetic kidney disease and related comorbidities, various specialist such as nephrologists, endocrinologists and primary care clinicians are collaborating.
2. Regular Monitoring:- To direct treatment and evaluate the progression of DKD requires frequent monitoring of blood pressure, glycaemic management (eGFR, UACR), renal function and other pertinent markers [46].

UACR measurements and GFR assessments should be less than 60ml/min per 1.73m² when diagnosed. It is within acceptable limits, even if GFR declines in situations of type 1 and type 2 diabetes. Diabetics must check their creatinine



levels annually. The MDRD formula or the CKD-EPI equation are being used to compute GFR. The screening for nephropathy in all patients with type 2 diabetes for at least five years has been recommended by the American Diabetes Association.

A tabular representation of the management and therapy of diabetic kidney disease (DKD).

1. Lipid Management:- Using statins to lower cardiovascular risk in people with DKD.

For example:- Atorvastatin, Rosuvastatin

2. Blood Pressure Management:- RAAS blocker use to lessen proteinuria and delay the development of DKD ARBs (eg. losartan), ACE inhibitors (eg. lisinopril)

3. Glycemic Control:- To show down the development of diabetic kidney disease strict management of glycaemic is must. For example:- SGLT2, Metformin and Insulin

4. RAAS Blockade:- Using ACE or ARBs inhibitors to maintain renal function.

For example:- Enalapril, Irbesartan.

5. Sodium Restriction:- Fluid retention and hypertension can be controlled by limiting the consumption of salt.

For example:- Dietary counseling

6. Protein Restriction:- Proteinuria can be reduced by restricting protein moderately.

For example:- Dietary counseling

7. Antiplatelet Therapy:- Antiplatelet medication used to lower cardiovascular risk.

For example:- Aspirin, Clopidogrel

8. Anticoagulation Therapy:- Anticoagulation has been into account in order to lower the risk of thromboembolism.

For example:- Enoxaparin, Apixaban

9. Regular Monitoring:- Regular Monitoring of Renal Function, blood pressure and glycaemic management should be done.

For example:- Blood pressure, UACR

10. Referral to Nephrology:- For thorough management, consultation is required with the nephrologist.

For example:-Nephrologist appointment

11. Lifestyle Modifications:- Healthy lifestyle has been encouraged and inclusion of healthy choices in life such as smoking cessation, Diet and exercise [47], [48].

CONCLUSION

As a result of continuous research into possible targets for prediction, monitoring and prevention. In recent years new and novel treatment has arisen in DKD. Nonetheless new developments are being incorporated into clinical practices through various makers. In future, to enhance doctors options for more effective and personalised therapies. Advances are being made in the creation of novel chemicals and therapeutic techniques. DKD is a medical calamity that has a high occuran roles, in addition there's a scarcity of new signs for early diagnosis and even the existing detection tools are proven ineffective. To assess the potential and applicability of novel plasma and urine biomarkers is required to have multinational and multicenter epidemiological studies. In recent years new treatments for diabetic kidney disease have progressed as a result of continuous search into various possible targets for monitoring



prediction and prevention of DKD. These incorporation of markers into clinical practices has made advances in the creation of more effective treatments and new therapeutic techniques are promised to enhance doctors choices in the identification and possible treatments in diabetic kidney disease.

REFERENCES

1. Hoogeveen EK. The epidemiology of diabetic kidney disease. *Kidney and Dialysis*. 2022;2(3):433–442. Available from: <https://doi.org/10.3390/kidneydial2030038>
2. Haller B, Ulm K, Hapfelmeier A. A simulation study comparing different statistical approaches for the identification of predictive biomarkers. *Computational and Mathematical Methods in Medicine*. 2019;2019:1–15. Available from: <https://doi.org/10.1155/2019/7037230>
3. Khurana N, James S, Coughlan MT, Macisaac RJ, Ekinici EI. Novel therapies for kidney disease in people with diabetes. *Journal of Clinical Endocrinology & Metabolism*. 2022;107(1):e1–e24. Available from: <https://doi.org/10.1210/clinem/dgab639>
4. Inker L, Coresh J, Levey A, Tonelli M, Muntner P. Estimated GFR, albuminuria, and complications of chronic kidney disease. *Journal of the American Society of Nephrology*. 2011;22(12):2322–2331. Available from: <https://doi.org/10.1681/asn.2010111181>
5. Picow E. Improving the identification and management of diabetic nephropathy in patients with diabetes in primary care. *Journal of the American Association of Nurse Practitioners*. 2023;35(11):740–746. Available from: <https://doi.org/10.1097/jxx.0000000000000921>
6. Kondhalkar AA, Hawale DV, Jha RK, Anjankar A. To study the potential biomarkers for the early identification of DKD in vidharbha region. *Bioscience Biotechnology Research Communications*. 2021;14(7):42–46. Available from: <http://dx.doi.org/10.21786/bbrc/14.7.10>
7. IDF Diabetes Atlas 2021 (10). International Diabetes Federation. 2021.
8. Sigfrids FJ, Groop PH. Progression and regression of kidney disease in type 1 diabetes. *Frontiers in Nephrology*. 2023;3:1–15. Available from: <https://doi.org/10.3389/fneph.2023.1282818>
9. Han Y, Lee K, Saha A, Han J, Choi H, Noh M, et al. Specialized proresolving mediators for therapeutic interventions targeting metabolic and inflammatory disorders. *Biomolecules & Therapeutics*. 2021;29(5):455–464. Available from: <https://doi.org/10.4062/biomolther.2021.094>
10. Yamazaki T, Mimura I, Tanaka T, Nangaku M. Treatment of diabetic kidney disease: current and future. *Diabetes & Metabolism Journal*. 2021;45(1):11–26. Available from: <https://doi.org/10.4093/dmj.2020.0217>
11. Deng Y, Li N, Wu Y, Wang M, Yang S, Zheng Y. Global, regional, and national burden of diabetes-related chronic kidney disease from 1990 to 2019. *Frontiers in Endocrinology*. 2021;12:1–15. Available from: <https://doi.org/10.3389/fendo.2021.672350>
12. Rico-Fontalvo J, Aroca-Martinez G, Daza-Arnedo R, Cabrales J, Yáñez T, Cardona-Blanco M, et al. Novel biomarkers of diabetic kidney disease. *Biomolecules*. 2023;13(4):1–13. Available from: <https://doi.org/10.3390/biom13040633>
13. Tamay-Cach F, Quintana-Pérez J, Trujillo-Ferrara J, Cuevas-Hernández R, Valle-Mondragón L, García-Trejo E. A review of the impact of oxidative stress and some antioxidant therapies on renal damage. *Renal Failure*. 2015;38(2):171–175. Available from: <https://doi.org/10.3109/0886022X.2015.1120097>
14. Inoguchi T, Sonta T, Tsubouchi H, Etoh T, Kakimoto M, Sonoda N, et al. Protein kinase C-dependent increase in reactive oxygen species (ROS) production in vascular tissues of diabetes: role of vascular NAD(P)H oxidase. *Journal of the American Society of*



- Nephrology. 2003;14(8 Suppl 3):S227–S232. Available from: <https://doi.org/10.1097/01.asn.0000077407.90309.65>
15. Inker L. Estimated glomerular filtration rate, albuminuria, and adverse outcomes. *JAMA*. 2023;330(13):1266–1277. Available from: <https://jamanetwork.com/journals/jama/article-abstract/2810033>
16. Hu H, Liang W, Zhang Z, Liu Z, FC, Bao Y, et al. The utility of perirenal fat in determining the risk of onset and progression of diabetic kidney disease. *International Journal of Endocrinology*. 2022;2022:1–10. Available from: <https://doi.org/10.1155/2022/2550744>
17. Reznichenko A, Nair V, Eddy S, Tomilo M, Slidel T, Ju W, et al. Molecular Stratification of Chronic Kidney Disease. *medRxiv*. 2021;p. 1–36. Available from: <https://doi.org/10.1101/2021.09.09.21263234>
18. Reinhardt W, Mülling N, Behrendt S, Benson S, Dolff S, Führer D, et al. Association between albuminuria and thyroid function in patients with chronic kidney disease. *Endocrine*. 2021;73(2):367–373. Available from: <https://doi.org/10.1007/s12020-021-02640-1>
19. Liao Y, Liao W, Liu J, Xu G, Zeng R. Assessment of the CKD-EPI equation to estimate glomerular filtration rate in adults from a chinese CKD population. *Journal of International Medical Research*. 2011;39(6):2273–2280. Available from: <https://doi.org/10.1177/147323001103900624>
20. Beben T, Rifkin DE. GFR estimating equations and liver disease. *Advances in Chronic Kidney Disease*. 2015;22(5):337–342. Available from: <https://doi.org/10.1053%2Fj.ackd.2015.05.003>
21. Bostom AG, Kronenberg F, Ritz E. Predictive Performance of Renal Function Equations for Patients with Chronic Kidney Disease and Normal Serum Creatinine Levels. *Journal of the American Society of Nephrology*. 2002;13(8):2140–2144. Available from: <https://dx.doi.org/10.1097/01.asn.0000022011.35035.f3>
22. Faubel S, Chawla LS, Chertow GM, Goldstein SL, Jaber BL, Liu KD. Ongoing Clinical Trials in AKI. *Clinical Journal of the American Society of Nephrology*. 2012;7(5):861–873. Available from: <https://dx.doi.org/10.2215/cjn.12191111>
23. Volpe M, Volpe R, Gallo G, Presta V, Tocci G, Folco E, et al. 2017 Position Paper of the Italian Society for Cardiovascular Prevention (SIPREC) for an Updated Clinical Management of Hypercholesterolemia and Cardiovascular Risk: Executive Document. *High Blood Pressure & Cardiovascular Prevention*. 2017;24(3):313–329. Available from: <https://dx.doi.org/10.1007/s40292-017-0211-6>
24. Abdallah NH, Kamel MF, Abdelaziz A, Elkareem RMA. Urinary Retinol Binding Protein (RBP) Level as an early Biomarker of Diabetic Nephropathy in type 2 Diabetic Patients. *Egyptian Journal of Medical Research*. 2020;1(2):49–60. Available from: <https://dx.doi.org/10.21608/ejmr.2020.90106>
25. Bonventre JV. Kidney Injury Molecule-1 (KIM-1): A specific and sensitive biomarker of kidney injury. *Scandinavian Journal of Clinical and Laboratory Investigation*. 2008;68(sup241):78–83. Available from: <https://dx.doi.org/10.1080/00365510802145059>
26. Lupusoru GE, Ailincal IG, Sorohan BM, Andronesi AG, Lupusoru M, Andronesi D, et al. Mo648 Serum soluble urokinase plasminogen activator receptor in patients with diabetic kidney disease. *Nephrology Dialysis Transplantation*. 2021;36(Supplement_1):1. Available from: <https://dx.doi.org/10.1093/ndt/gfab094.0016>
27. Kamijo-Ikemori A, Sugaya T, Ichikawa D, Hoshino S, Matsui K, Yokoyama T, et al. Urinary liver type fatty acid binding protein in diabetic nephropathy. *Clinica Chimica Acta*. 2013;424:104–108. Available from: <https://dx.doi.org/10.1016/j.cca.2013.05.020>
28. Juretzko A, Steinbach A, Hannemann A, Endlich K, Endlich N, Friedrich N, et al.



- Urinary Angiotensinogen and Renin Excretion are Associated with Chronic Kidney Disease. *Kidney and Blood Pressure Research*. 2017;42(1):145–155. Available from: <https://dx.doi.org/10.1159/000474932>
29. Wen CP, Chang CH, Tsai MK, Lee JH, Lu PJ, Tsai SP, et al. Diabetes with early kidney involvement may shorten life expectancy by 16 years. *Kidney International*. 2017;92(2):388–396. Available from: <https://dx.doi.org/10.1016/j.kint.2017.01.030>
 30. Lecamwasam A, Ekinci E, Dwyer K, Saffery R. The identification of potential biomarkers of chronic kidney disease in individuals with diabetes. *Kidney International Reports*. 2019;4(7):1. Available from: <https://doi.org/10.1016/j.ekir.2019.05.1100>
 31. Ali SI, Morsy AA, Mohammed RA, Gelany HAE. Kim-1 and NGAL as biomarkers of nephropathy in type II diabetes. *International Journal of Advanced Research*. 2018;6(2):1412–1417. Available from: <http://dx.doi.org/10.21474/IJAR01/6571>
 32. Perco P, Pena M, Heerspink H, Mayer G. Multimarker panels in diabetic kidney disease: the way to improved clinical trial design and clinical practice. *kidney International Reports*. 2019;4(2):212–221. Available from: <https://doi.org/10.1016%2Fj.ekir.2018.12.001>
 33. Tonelli M, Muntner P, Lloyd A, Manns BJ, Klarenbach S, Pannu N, et al. Risk of coronary events in people with chronic kidney disease compared with those with diabetes: a population-level cohort study. *The Lancet*. 2012;380(9844):807–814. Available from: [https://dx.doi.org/10.1016/s0140-6736\(12\)60572-8](https://dx.doi.org/10.1016/s0140-6736(12)60572-8)
 34. Kourtidou C, Stangou M, Marinaki S, Tziomalos K. Novel Cardiovascular Risk Factors in Patients with Diabetic Kidney Disease. *International Journal of Molecular Sciences*. 2021;22(20):1–11. Available from: <https://doi.org/10.3390%2Fijms222011196>
 35. Hussain S, Jamali M, Habib A, Hussain S, Akhtar M, Najmi A. Diabetic kidney disease: an overview of prevalence, risk factors, and biomarkers. *Clinical Epidemiology and Global Health*. 2021;9:2–6. Available from: <https://doi.org/10.1016/j.cegh.2020.05.016>
 36. Gameil MA, Marzouk RE, Elsebaie AH, Eldeeb AAA. Albuminuria independently determines intact parathyroid hormone in diabetic nephropathy with micro-albuminuria. *The Egyptian Journal of Hospital Medicine*. 2021;84(1):2052–2056. Available from: <https://dx.doi.org/10.21608/ejhm.2021.180351>
 37. Lisowska-Myjak B. Serum and urinary biomarkers of acute kidney injury. *Blood Purification*. 2010;29(4):357–365. Available from: <https://doi.org/10.1159/000309421>
 38. Kumar GS, Sandhya S, Tejaswini T, Raghavendra B. A review on biomarkers for early diagnosis of diabetic kidney disease. *International Journal for Multidisciplinary Research*. 2023;5(1):1–4. Available from: <https://doi.org/10.36948/ijfmr.2023.v05i01.1345>
 39. Levey AS, Becker C, Inker LA. Glomerular filtration rate and albuminuria for detection and staging of acute and chronic kidney disease in adults. *JAMA*. 2015;313(8):837–846. Available from: <https://doi.org/10.1001/jama.2015.0602>
 40. García-Carro C, Vergara A, Bermejo S, Azancot MA, Sánchez-Fructuoso AI, Nieta MDSdl, et al. How to assess diabetic kidney disease progression from albuminuria to GFR. *Journal of Clinical Medicine*. 2021;10(11):1–13. Available from: <https://doi.org/10.3390%2Fjcm10112505>
 41. Skolnik NS, Style AJ. Importance of early screening and diagnosis of chronic kidney disease in patients with type 2 diabetes. *diabetes Therapy*. 2021;12(6):1613–1630. Available from: <https://doi.org/10.1007/s13300-021-01050-w>
 42. Erman A, Rahamimov R, Mashraki T, Levy-Drummer R, Winkler J, DI. The Urine Albumin-to-Creatinine Ratio: Assessment of Its Performance in the Renal Transplant Recipient Population. *Clinical Journal of the American Society of Nephrology*.



- 2011;6(4):892–897. Available from: <https://doi.org/10.2215%2FCJN.05280610>
43. Bukabau J, Yayo E, Gnionsahé A, Monnet D, Pottel H, Cavalier É, et al. Performance of creatinine- or cystatin c-based equations to estimate glomerular filtration rate in sub-saharan african populations. *Kidney International*. 2019;95(5):1181–1189. Available from: <https://doi.org/10.1016/j.kint.2018.11.045>
 44. Sontrop JM, Garg AX, Li L, Gallo K, Schumann V, Winick-Ng J, et al. Consecutive first-morning urine samples to measure change in the albumin-to-creatinine ratio: a pilot study of a home urine collection protocol. *Canadian Journal of Kidney Health and Disease*. 2016;3:1–8. Available from: <https://doi.org/10.1186/s40697-016-0095-8>
 45. Ebert N, Bevc S, Bökenkamp A, Gaillard F, Hornum M, Jager KJ, et al. Assessment of kidney function: clinical indications for measured gfr. *Clinical Kidney Journal*. 2021;14(8):1861–1870. Available from: <https://doi.org/10.1093%2Fckj%2Fsfab042>
 46. Looker HC, Mauer M, Nelson RG. Role of kidney biopsies for biomarker discovery in diabetic kidney disease. *Advances in Chronic kidney Disease*. 2018;25(2):192–201. Available from: <https://doi.org/10.1053%2Fj.ackd.2017.11.004>
 47. Koyner JL, Vaidya VS, Bennett MR, Ma Q, Worcester E, Akhter SA, et al. Urinary biomarkers in the clinical prognosis and early detection of acute kidney injury. *Clinical Journal of the American Society of Nephrology*. 2010;5(12):2154–2165. Available from: <https://doi.org/10.2215/cjn.00740110>
 48. Roberts I, Mason P, Fogo A. The renal biopsy . In: *Oxford Textbook of Clinical Nephrology*. (pp. 142-160) Oxford University Press. 2015.

HOW TO CITE: Yuvraj Rattu, Rajesh Kumar, Ajeet Pal Singh, Amar Pal Singh, Gaurav Hastir, Review on Diabetic Kidney Disease, *Int. J. of Pharm. Sci.*, 2026, Vol 4, Issue 9, 1229-1241. <https://doi.org/10.5281/zenodo.22704747>

