Indian Journal of Health Care, Medical & Pharmacy Practice Vol 6; Issue 1, Jan-Jun 2025, ISSN 2583-2069

Review Article

DIABETIC KIDNEY DISEASE: PREVALENCE, DIAGNOSIS, RISK FACTORS AND TREATMENT

Dr. Mosim[1]*, Dr. Bhupendra Kumar Kumawat[2], Ashutosh Kumar[3], Liyakat Ali[4], Aarif Khan[5]

[1]Assistant Professor, [2,3,4,5]Department of Pharmacy Practice, Northern Institute of Pharmacy & Research, Alwar, Rajasthan, India.

Corresponding Author*: Dr. Mosim, Assistant Professor, Department of Pharmacy Practice, Northern Institute of Pharmacy & Research, Alwar, Rajasthan, India.

Email ID: mosimk9@gmail.com

DOI: https://doi.org/10.59551/IJHMP/25832069/2025.6.1.20

Received: 11 Jan, 2025, Decision for Acceptance: 16 Feb, 2025

Abstract

Diabetes: Diabetic kidney disease (DKD) is recognized as the predominant ethology of end-stage kidney disease globally and represents the most critical prognostic factor for mortality in patients with diabetes. Diabetic kidney disease affects approximately 20% of the 400 million people worldwide who have diabetes mellitus (DKD). DKD is associated with increased cardiovascular and all-cause morbidity and mortality, so early detection and treatment are critical. The optimal method in order to identify DKD early involves conducting an annual spot urine albumin-to-creatinine ratio test, with the diagnosis confirmed by consistent elevations in urinary albumin excretion upon repeat testing. The pathophysiology of DKD involves multiple mechanisms, including processes related to metabolism, inflammation, and hemodynamic. Hyperglycaemia-induced activation of the electron transport chain leads to a rise in ROS (Reactive Oxygen Species), which is considered a key initiator of diabetes-related complications. Several antihyperglycemic agents, including dipeptidyl peptidase-4 (DPP-4) inhibitors, glucagon-like peptide-1 (GLP-1) receptor agonists, and sodium-glucose cotransporter-2 (SGLT-2) inhibitors, may help prevent the onset and progression of DKD by effectively lowering blood glucose levels and offering inherent renal protection. To prevent microvascular complications, it is essential to monitor blood pressure during each clinical visit and maintain it below 140/90 mm Hg. This article seeks to the patients advancing referring patients to nephrogenic subspecialists may be beneficial if they have stage 3 DKD or more due to the disease's complexity and the increased risk of adverse outcomes.

Keywords: DKD, Urine Albumin, Hemodynamic, Metabolic, Hyperglycaemia, Antihyperglycemic, Microvascular

1. Introduction

Diabetes mellitus affects over 400 million people worldwide, with nearly 600 million anticipated to have an impact by 2035. In the US, diabetes affects about 12% of the population, and up to 25% of those individuals do not receive a diagnosis. Patients of all ages, genders, income levels, racial or ethnic groups, and educational attainment are affected by the disease. Diabatic Kidney Disease (DKD) affects approximately 20% of diabetic patients. DKD has an association with increased morbidity and mortality

risks, and it is the major reason of End-Stage Renal Disease (ESRD) among the Americans [1].

Preventing diabetes in the general population is the most effective strategy to mitigate the burden of DKD. By identifying and understanding the key risk factors for DKD development, healthcare providers can facilitate early detection and timely interventions, potentially reducing the progression and severity of the disease. Reducing the prevalence of diabetes in the populace at large is the best performing approach to decreasing the DKD's effect. Identifying and understanding risk elements for the onset of DKD can enable earlier detection and more targeted interventions, thereby slowing disease progression and improving patient outcomes. Effective use of screening protocols, therapeutic approaches, and referrals to subspecialties can aid in the prevention of DKD progression. The role Primary care doctors' involvement in the treatment of individuals with type 2 diabetes who have DKD is discussed [2].

2. Risk Factors of DKD

2.1 Increased Albuminuria

Elevated urinary albumin excretion is a key risk element for renal disease's initiation and development in individuals with diabetes. Microalbuminuria (30–300 mg/g) or macroalbuminuria (> 300 mg/g) is characterised by increased excretion of albumin/g creatinine in the urine.

2.2 Hyperglycaemia

Hyperglycaemia is regarded among the many significant and independent risk factors for DKD. It worsens renal function by altering the antioxidant system, resulting in increased creation of final goods with advanced glycation. The activation pertaining to the polyol pathway is believed to additionally be implicated in DKD's pathogenesis. Variability in glycated haemoglobin (HbA1c) is connected to the growth and advancement of nephropathy in both individuals with type 1 and type 2 diabetes.

2.3 Smoking

Tobacco use is considered a separate risk element for both the onset and progression of diabetic nephropathy. Smoking plays a multifactorial part in the onset of Diabetic Kidney Disease (DKD), contributing through mechanisms such as oxidative stress, hyperlipidaemia, deposition of end products related to Advanced Glycation (AGEs), and glomerulosclerosis. Hypertension Diabetes poses a significant danger for nephropathy caused by diabetes. A recent meta-analysis confirmed that the onset of diabetic nephropathy is substantially linked to hypertension. Hypertension is linked to cardiovascular disease in kids with CKD. Patients with hypertension have greater likelihood of developing diabetic nephropathy than those without the condition.

Pathophysiology of DKD

DKD pathophysiology involves several routes, such as mechanisms related to hemodynamic, metabolism, and inflammation. The polyol, hexosamine, advanced glycation end products (AGEs), and protein kinase C pathways are all integral components of the metabolic pathway involved in diabetic kidney disease (DKD). 46 Hyperglycaemia contributes to kidney damage via the hemodynamic route by increasing the production of end products that are glycosylated, activating protein kinase C (PKC), and promoting diacylglycerol synthesis. 47 The formation of reactive oxygen species (ROS) induced by hyperglycaemia, primarily through the activation of the electron transport chain, is said to be a key contributor to the onset of diabetes complications[3].

Diagnosis

DKD is diagnosed by measuring the Glomerular Filtration Rate (GFR) and Urinary Albumin Concentration Rate (UACR) for testing albumin presence in urine. It is clinically identified by an estimated GFR of less than 60 ml/min per1.73 m2 or persistent albumin excretion in the urine (albuminuria). Despite having a normal UACR,

GFR decreased in both T1DM and T2DM. As a result, creatinine levels in diabetic patients must be measured on an annual basis. There are several equations for calculating estimated GFR, such as the Chronic Kidney Epidemiology Collaboration (CKD-EPI) equation, which takes into account weight, transplant, and diabetes as potential variables.

The Modified Diet in Renal Disease (MDRD) equation, which computes GFR based on creatinine levels in the serum and patient characteristics, is another commonly used equation. The CKD-EPI equation outperformed the MDRD equation in accurately stratifying CKD risk[4].

DKD biomarkers for early detection Early diagnosis and timely intervention are considered the most effective strategies for managing diabetic kidney disease (DKD). Detecting DKD at an early stage allows for interventions that can slow progression, including the control of blood glucose, blood pressure, and albuminuria. Early detection can have long-term benefits by slowing disease progression, increasing life expectancy, and lowering the economic and humanistic burden. Currently, serum creatinine and albuminuria are the only clinically available markers for identifying DKD[5].

Treatment

The identification of macroalbuminuric patients enables early intervention to prevent the course of Diabetic Kidney Disease (DKD) and lower the chance of developing End-Stage Renal Disease (ESRD). DKD treatment primarily entails cautious control of high blood pressure and blood sugar levels, along with the utilization of drugs that provide specific renal benefit. Additional risk variables that may be changed should also be considered.

2.4 Glycaemic Control

Although no significant studies have examined the best glycaemic goals to avoid DKD, numerous research has attempted to elucidate the ideal degree of glycaemic control in order to avoid diabetes-related microvascular (e.g., DKD, retinopathy) problems and macrovascular (e.g. stroke, mortality, myocardial infarction). Hyperglycaemia ought to be handled with a multifaceted approach that includes weight loss, exercise, dietary changes, and medication. Metformin and lifestyle modifications continue to be the first-line therapy for diabetic individuals[6].

2.4 Blood Pressure Control

Controlling Blood Pressure (BP) is critical for preventing and slowing the development of DKD. A routine blood pressure examination should be part of every clinical appointment. 32 The standard protocols for diagnosing and monitoring blood pressure should be followed (encompassing the patient sit with his or her arms in support, feet on the ground, and ambulatory blood pressure monitoring).

To reduce the prevalence of DKD and other microvascular diseases, ideal blood pressure is maintained below 90 mm Hg for the diastolic and below 140 mm Hg for the systolic. 35 For certain cases (e.g. people with confirmed DKD or other elevated risk factors for Atherosclerotic Cardiovascular Disease) low aims (130/80 mm Hg) could be if achieved with minimal treatment load or adverse effects[7].

2.5 Lipids Management

DKD modifies metabolism of lipids, resulting in an increase in the low-density lipoprotein-cholesterol complex and an increased chance of negative consequences. associated with atherosclerotic cardiovascular disease. While statin therapy has no effect on DKD progression, it does reduce patient's mortality and cardiac events in individuals with renal illness that is not dependent on dialysis (both without and with diabetes). 5 Because many statins are metabolised by the kidneys, dosages ought to be decreased if patient's eGFR is significantly lower. The doses of atorvastatin (Lipitor) don't require modification[8].

2.6 Dietary Modification

Dietary changes have the potential to slow down the DKD evolution; nevertheless, the data supporting

particular therapies is conflicting. According to the American Diabates Association, in DKD's patients, this can lower GFR progression and reduction.to ESRD. A diet that is Mediterranean and DASH (Dietary Approaches to Stop Hypertension) diet can both be beneficial. Whole-grain carbohydrates, fibre, omega-3 and omega-9 fats, fresh produce, and fewer than 2,300 mg of sodium per day are all part of these diets. Sugary foods, processed carbs and saturated fats ought to be prevented. 49 Routine monitoring for changes in vitamin D, potassium and phosphorus levels in DKD patients may direct further dietary adjustments[9].

3. Conclusion

Due to its high prevalence, inadequate diagnosis, and lack of new biomarkers for early detection, DKD is a medical catastrophe. The early detection of Diabetic Kidney Disease (DKD) may be greatly aided by newly developed plasma and urine biomarkers. These biomarkers, which include markers like urine NGAL, KIM-1, and urinary cystatin C, could serve as cost-effective diagnostic tools to identify DKD at an earlier stage. However, in order to validate the potential and applicability of these biomarkers, multination and multicentre epidemiological studies are required as an urgent task.

4. Conflict of Interest: None

5. References

1. Y. Chen, K. Lee, Z. Ni, and J. C. He, "Diabetic kidney disease: Challenges, advances, and opportunities," Kidney Diseases, vol. 6, no. 4, pp. 215–225, 2020.
2. R. Z. Alicic, M. T. Rooney, and K. R. Tuttle, "Diabetic kidney disease: Challenges, progress, and possibilities," Clinical Journal of the American Society of Nephrology, vol. 12, no. 12, pp. 2032–2045, 2017.
3. S. Hussain, M. C. Jamali, A. Habib, M. S. Hussain, M. Akhtar, and A. K. Najmi, "Diabetic kidney disease: An overview of prevalence, risk factors, and biomarkers," Clinical Epidemiology and Global Health, vol. 9, pp. 2–6, 2021.
4. K. Reidy, T. Hostetter, and K. Susztak, "Molecular mechanisms of diabetic kidney disease," Journal of Clinical Investigation, vol. 124, no. 6, pp. 2333–2340, 2014.
5. E. C. Wylie and S. C. Satchell, "Diabetic nephropathy," Clinical Medicine, vol. 12, no. 5, pp. 480–482, 2012.
6. K. McGrath and R. Edi, "Diabetic kidney disease: Diagnosis, treatment, and prevention," Elsevier, vol. 8, no. 7, pp. 78–93, [Year].
7. N. M. Selby and M. W. Taal, "An updated overview of diabetic nephropathy: Diagnosis, prognosis, treatment goals, and latest guidelines," Diabetes, Obesity and Metabolism, vol. 22, pp. 3–15, 2020.
8. N. Samsu, "Diabetic nephropathy: Challenges in pathogenesis, diagnosis, and treatment," BioMed Research International, vol. 2021, Article ID 1497449, 2021.
9. S. P. Srivastava, K. Kanasaki, and J. E. Goodwin, "Editorial: Combating diabetes and diabetic kidney disease," Frontiers in Pharmacology, vol. 12, Article ID 716029, 2021.

Cite this article Mosim et al., Diabetic Kidney Disease: Prevalence, Diagnosis, Risk Factors and Treatment. Indian Journal of Health Care, Medical & Pharmacy Practice. 2025;6(1):11-14.