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*Review Article*

# Chronic Kidney Disease: A Comprehensive Review

**Utkarsh Kumar*[1], Shashi Bhushan[2], Ritul Kumari[1], Sneha Kumari[1]**

[1]*Muzaffarpur College of Professional Education.*
[2]*Nibha Institute of Pharmaceutical Sciences Rajgir Nalanda Bihar*

Chronic kidney disease (CKD) decreases the renal function. It is also known as chronic renal disease and can be life-threatening. CKD causes various diseases, including hypertension, anemia, rashes, muscle pain, and conjunctivitis. Hypertension and diabetes are the main causes of chronic kidney disease. In this disease, the Glomerular Filtration Rate (GFR) decreases. Patients with chronic kidney disease have various metabolic abnormalities, such as electrolyte imbalance and uremia (urine in the blood). This review discusses the introduction, epidemiology, pathophysiology, diagnosis, and future prospects of this condition.

**Keywords**: Chronic Kidney diseases, GFR (Glomerular Filtration Rate).

## INTRODUCTION

The kidneys are bean-shaped organs. Kidney typically weighs between 120 to 200g. These data vary between men and women. In males, the brain weighs 160–200 g, whereas in females, it weighs 130–140 g. The kidneys are comparable in size to a closed fist. Chronic kidney disease is one of the most common causes of death. In this disease, patients are affected by abnormalities in kidney function. Various symptoms, including poor appetite, foot swelling, and muscle cramps, were observed. CKD is a progressive disorder characterized by irreversible loss of kidney structure and function. The Kidney is a vital organ responsible for maintaining homeostasis, electrolyte balance, and blood pressure. Early detection and management of chronic kidney diseases can reduce complications, such as kidney failure GFR value increased GFR. Pharmacological approaches to manage chronic kidney diseases include controlling glycemic levels, blood pressure, and proteinuria (abnormal excessive amount of protein leaking into the urine). This review provides a comprehensive overview of chronic kidney, focusing on its pathophysiology, etiology, and current treatment approaches.

| Stage | eGFR | What it means |
|---|---|---|
| Stage-1 | 90 or higher | Mild Damage; Kidney work as normal |
| Stage-2 | 60-89 | Mild Damage; Kidney still work |
| Stage-3 | 45-59 | Mild to Moderate Damage; Kidneys don't work well as they should |
| Stage-4 | 30-44 | Severe Damage; Kidneys are close to not work at all |
| Stage-5 | Less than 15 | Most Severe Damage; Kidneys are close to not working or stop working |

### Epidemiology

Chronic kidney disease is a long-term progressive condition characterized by a gradual and irreversible decline in kidney function. CKD is a major public health challenge worldwide because it remains undiagnosed until it reaches an advanced stage of the disease. Chronic kidney disease affects globally nearly 10-13% of the global adult population. It Increases due to the growing incidence of hypertension, diabetes, and an aging population. Chronic kidney diseases in stages 1 and 2 are the most

common at the population level; however, they are usually asymptomatic and often undiagnosed. In stages 3 and 4, patients begin to experience clinical symptoms such as fatigue, edema, and anemia. Chronic Kidney disease is observed in middle-aged and elderly individuals, with the risk increasing after the age of 40 years. The epidemiology of CKD is linked to diabetes, hypertension, and cardiovascular diseases.

**Etiology**

The causes of chronic kidney diseases are condition like the

1. Diabetes Mellitus
2. Hypertension
3. Primary Glomerulonephritis

**1. Diabetes Mellitus**

Diabetes Mellitus is a major cause of kidney disease worldwide. In CKD, hyperglycemia induces structural and functional alterations in the kidneys, resulting in kidney hyperfiltration. This type of change eventually leads to diabetic nephropathy, decreases the glomerular filtration rate, and increases creatinine levels in the body, which can cause severe complications.

**2. Hypertension**

Chronic hypertension is also a major cause of the chronic kidney disease. Blood pressure increased. It damages blood vessels, causing nephrosclerosis and nephron dysfunction.

**3. Chronic Infection**

In some cases, untreated urinary tract infections can cause renal issues, and chronic inflammation can lead to complications such as increased creatinine levels in the body, which can cause renal issues.

**4. Drug induced nephrotoxicity**

Some types of non-steroidal anti-inflammatory drugs (NSAIDs) can cause kidney damage with long-term or excessive use.

**Pathophysiology Of CKD**

Chronic Kidney disease (CKD) is associated with a progressive and irreversible decline in renal system function, leading to long-term structural and functional damage to the kidneys. Starting with the initial cause, the progression of chronic kidney disease occurs after a scenario called renal fibrosis. Various risk factors, such as diabetes mellitus, hypertension, chronic glomerulonephritis, and other inflammatory renal responses, initiate repeated injuries to renal tissues, ultimately leading to chronic scarring and loss of functional nephrons. Renal fibrosis is indicative of an abnormal wound-healing response to consistent injury and is the core mechanism underlying CKD progression. In addition to restoring the renal structure, consistent injury results in excessive deposition of the extracellular matrix, leading to glomerulosclerosis, tubular atrophy, and interstitial fibrosis. These structural changes rapidly reduce the filtering capacity of the kidneys and degrade renal functions. Renal fibrosis can result from diverse renal diseases that progress to end-stage kidney disease (ESKD). Injured renal cells by renal fibrosis release chemokines and cytokines, attracting inflammatory cells such as macrophages, monocytes, lymphocytes, and neutrophils to the damaged site. These cells accumulate in the glomeruli and interstitium and release inflammatory mediators, promoting tissue injury and fibrosis in the kidneys. Consistent inflammation plays an important role in CKD progression. Disruption of the glomerular basement membrane structure and podocyte injury are considered the second phase of CKD progression. Podocyte damage leads to the breakdown of foot processes and disruption of the slit diaphragm, resulting in increased glomerular permeability to plasma proteins. Thus, proteins (particularly albumin) are abnormally filtered and excreted in the urine, causing proteinuria. Proteinuria is not only a part of CKD progression but also plays an important role in long-term renal injury. Excessive protein filtration increases the protein pressure in the renal tubules. The reabsorption of these proteins by tubular epithelial cells leads to cellular stress and inflammation. Long-term protein uptake can cause tubular injury, apoptosis, tubular atrophy, interstitial inflammation, and fibrosis. The continuity of tubular injury and interstitial fibrosis reduces oxygen levels, impairs tubular function, and leads to nephron loss. Over time, these factors can collectively lead to a decrease in the

glomerular filtration rate, worsen renal function, and result in end-stage renal disease (ESRD).

**Clinical Manifestations and Complications of CKD**

The clinical manifestations of chronic kidney disease (CKD) develop over time as renal function degrades and progresses to the uremic stage. These manifestations are commonly divided into primary (renal) and secondary (systemic) features of the disease.

**1. Primary manifestations**

Primary renal manifestations occur due to the consistent loss of nephron function, causing disturbances in the fluid, electrolyte, and acid-base balance.

- Metabolic acidosis occurs when the kidneys are unable to excrete hydrogen ions and regenerate bicarbonate, resulting in symptoms such as deep breathing (Kussmaul respiration) and fatigue. Chronic acidosis contributes to bone demineralization, muscle protein breakdown, and kidney disease progression.

- **Hyperkalemia:** Hyperkalemia develops due to reduced potassium excretion, which may lead to cardiac arrhythmia and muscle weakness.

- **Sodium and Water Imbalance-** The Sodium and water imbalance occurs due to a decrease in GFR, which causes retention of salt and water, producing hypervolemia, edema, hypertension, and eventually congestive heart failure.

- **Azotemia:** Due to the reduced excretion of nitrogenous waste products, azotemia may occur, which is characterized by elevated blood levels of urea and creatinine, causing toxic effects and contributing to uremic symptoms. Hyperuricemia may occur with the deposition of urate crystals in the joints and soft tissues.

**2. Secondary manifestations and Complications**

As kidney function continues to decline, disturbances occur in multiple body systems, resulting in various secondary clinical manifestations and complications.

- **Anemia:** Anemia is one of the earliest and most common complications of CKD. It occurs due to decreased erythropoietin production by the affected kidney. Anemia is commonly caused by a reduced red blood cell count and blood loss. Uremia-induced inhibitors of erythropoiesis, shortened red blood cell survival, and iron deficiency (from excess hepcidin impairing dietary absorption or functional iron deficiency from reticuloendothelial cell iron blockade) can also contribute to CKD anemia. Anemia in CKD is associated with poor outcomes, including reduced quality of life, increased incidence of cardiovascular disease, higher rates of hospital admissions, cognitive impairment, and mortality.

- **Cardiovascular disease (C)** is the leading cause of morbidity and mortality in patients with CKD, and the prevalence and burden of this complication increase with declining kidney function. Cardiovascular diseases are common and include hypertension, fluid overload, left ventricular hypertrophy, coronary artery disease, heart failure, and uremic pericarditis with effusion. These complications arise due to volume expansion, accelerated atherosclerosis, and the toxic effects of retained uremic substances.

- **Respiratory disease:** CKD does not directly cause primary lung disease; however, respiratory manifestations occur secondary to systemic disturbances. Fluid overload can lead to pulmonary edema, resulting in dyspnea. In addition, metabolic acidosis stimulates compensatory hyperventilation, resulting in Kussmaul respiration. Patients with advanced CKD are more susceptible to respiratory infections because of impaired immunity.

- **Neurological Complications:** Accumulation of uremic toxins and electrolyte imbalances adversely affect the nervous system. Neurological manifestations include cognitive impairment, difficulty concentrating, peripheral

neuropathy, restless leg syndrome, and asterixis. In severe cases, patients may develop seizures and uremic encephalopathy. These complications significantly impair quality of life and daily functioning.

- **Gastrointestinal Manifestations-** Gastrointestinal symptoms are common in CKD due to the toxic effects of retained metabolic waste products. Patients may experience nausea, vomiting, anorexia, weight loss, altered taste sensation (dysgeusia), and uremic fetor, which is characterized by an ammonia-like breath odor. In advanced stages, gastritis and gastrointestinal bleeding may occur, contributing to malnutrition and poor clinical outcomes.

Mineral bone disease is a common complication of CKD and can present as any combination of abnormalities in calcium, phosphate, parathyroid hormone (PTH), or vitamin D metabolism, which are usually recognized on abnormal biochemistry tests, such as increased serum phosphate and PTH concentrations, while the amount of serum calcium might be low, normal, or increased; abnormalities in bone turnover, mineralization, growth, or strength, which can manifest as bone pain or increased bone fragility; or extraskeletal calcification (including blood vessels and skin). As CKD progresses, active vitamin D deficiency increases, resulting in hypocalcemia and secondary (and eventually tertiary) hyperparathyroidism, leading to stimulation of bone osteoclast activity.

**Diagnosis of Chronic Kidney Disease**

The diagnosis of chronic kidney disease (CKD) is complex because it involves the progressive loss of nephron function and gradual degeneration of renal tissues. Owing to the slow development of CKD, it often remains undetected until significant kidney damage has occurred. Therefore, precise and early diagnosis is important to reduce the disease burden, slow disease progression, and improve the patients' quality of life. Diagnosis is based on a combination of traditional laboratory indicators, emerging biomarkers, and advanced imaging modalities, all of which contribute to the overall clinical assessment of kidney structure and function. Traditional markers remain central to the detection and diagnosis of CKD because they provide valuable information on kidney function and disease progression. A reduction in kidney function is often the earliest detectable indicator, with early stages (stages 1–3) providing an opportunity for timely opportunity to limit disease progression and complications. The last stages (Stages 4 and 5) are related to extensive renal impairment and can progress rapidly to end-stage kidney disease. Clinical manifestations may vary widely and are not always correlated with the severity of kidney injury, which is accurately diagnosed through investigations such as renal biopsies. Although biopsies are invasive and costly, there is a growing interest in alternative diagnostic approaches. Routine clinical evaluation involves laboratory assessment of serum creatinine and estimation of the glomerular filtration rate (eGFR), which shows the kidney's ability to excrete metabolic waste products. In addition to standardization, serum creatinine-based eGFR calculations can be inaccurate because of non-GFR factors. In these cases, cystatin C is a useful alternative marker, specifically for the prognostic monitoring and confirmation of kidney dysfunction in chronic disease. Recent advancements have introduced several innovative biomarkers that improve the sensitivity and precision of CKD diagnosis in patients with diabetes mellitus. In addition, the urinary albumin-to-creatinine ratio (UACR) is a strong predictor of kidney damage and cardiovascular risk. It can detect even minimal increases in albumin in urine, making it significantly valuable for early detection and monitoring of disease progression, often earlier than any particular changes in eGFR. Neutrophil gelatinase-associated lipocalin (NGAL) have also gained attention as a biomarker, with urinary NGAL showing good correlation in early kidney damage. Kidney injury molecule-1 (KIM-1), which is expressed in proximal tubular cells following ischemic or toxic injury, is another sensitive indicator of early stage renal injury and can serve as a marker for chronic renal disease caused by tubulointerstitial injury. In addition, symmetric dimethylarginine (SDMA), a stable metabolic byproduct excreted primarily by the kidneys, has been recognized as a reliable marker of renal function because of its low biological variability and minimal influence of non-renal factors. Proteomic approaches enhance diagnostic accuracy by identifying disease-specific protein fragments in urine, enabling the early

detection of pathological changes compared with conventional markers such as creatinine and albumin. Imaging modalities play an important role in the diagnosis of CKD by providing structural and anatomical information regarding the kidneys. Techniques such as ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) help evaluate the kidney size, morphology, cysts, stones, and obstructive lesions. Ultrasonography is frequently the first-line imaging tool because of its accessibility and lack of radiation exposure; however, CT and MRI provide a higher detection ability for detailed structural assessment. MRI is particularly useful when CT findings are inconclusive, or contrast exposure is contraindicated. Despite their diagnostic value, imaging techniques must be selected wisely to balance the benefits and potential risks, especially radiation exposure. Incorporating imaging findings with laboratory markers and emerging biomarkers enables a comprehensive approach to the diagnosis of CKD. As diagnostic technologies continue to evolve, they rely on greater accuracy, earlier detection, and improved clinical outcomes through personalized and preventive management.

**Current Treatment Approach For CKD**

As CKD continues to increase worldwide, along with the demand for related life-saving therapies, the financial burden of CKD will increasingly deplete the healthcare systems. Experimental studies have shown that glomerular capillary hypertension and impaired filtration, which result in protein overload, play significant roles in CKD progression. Consistently, human studies have shown that proteinuria is an independent predictor of disease progression, and its reduction is renoprotective in nature. At comparable BP control, inhibitors of the renin-angiotensin system (RAS), including angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), are more effective than non-RAS inhibitor therapy in reducing proteinuria, slowing the progression to ESRD, and even improving kidney function, achieving disease regression in some cases. In participants with diabetes, RAS inhibitors delay the onset of microalbuminuria and its progression to macroalbuminuria, and ACE inhibitors may reduce the excess cardiovascular mortality associated with diabetic kidney disease. However, the optimal management of CKD often requires a multimodal approach, in addition to RAS inhibition. Lifestyle modifications and combination drug therapy are necessary to control the multiple risk factors contributing to CKD progression and the development of cardiovascular complications in patients with CKD. The potential role of newer medications in further improving the effectiveness and cost-efficiency of renoprotective strategies remains an active area of research. Among 360 nondiabetic CKD patients included in the Renoprotection of Optimal Antiproteinuric Doses (ROAD) study, those randomized to 3.7-year treatment with benazepril or losartan at a dose that was uptitrated until the maximum antiproteinuric effect had a reduced incidence of the combined endpoint of doubling of serum creatinine, ESRD, or death compared with those on conventional antihypertensive doses of both medications. However, the effects of benazepril and losartan on the considered outcomes were similar, regardless of the administered treatment dose or duration. In apparent harmony with the above findings, the authors of the Diabetics Exposed to The Telmisartan and Enalapril (DETAIL) trial concluded that, based on predefined criteria, telmisartan was not less effective than enalapril in 250 type 2 patients with diabetes, hypertension, and micro- or macroalbuminuria. One of the most promising novel drugs is the renin inhibitor aliskiren, which was found to significantly reduce albuminuria compared to placebo in 599 patients with type 2 diabetes and nephropathy who received background therapy with losartan. Renin inhibition is one of the mechanisms that have been suggested to support the antiproteinuric effect of the vitamin D receptor agonist, paricalcitol. In a short-term trial in albuminuric patients with diabetes on background ARB therapy, this drug significantly reduced albuminuria; however, the results were confounded by the lower BP in the active treatment group than that in the placebo group. In a broad range of animal models of proteinuric kidney disease, ACE inhibitors, ARBs, or both not only prevented progressive renal damage but also induced the regression of glomerulosclerosis, tubulointerstitial lesions, and vascular lesions. A long-term follow-up of the REIN study showed that the rate of measured GFR decline progressively improved to a level of

approximately 1 ml/min per 1.73 m² per year after at least 5 years of continued ramipril use, which approximates the average age-related loss in GFR over time in healthy participants. Moreover, a breakpoint was identified in the slope of GFR changes over time, which started to increase after 36 months of treatment, leading to the hypothesis that renal disease can be reversed. The integrated use of different treatments against the same target, such as uncontrolled cell or viral replication, has dramatically improved the outcomes of severe diseases, such as cancer and acquired immunodeficiency syndrome (AIDS). By analogy, a multimodal intervention strategy using all available tools to target a major pathogenic factor in the progression of CKD, such as proteinuria, seems to be a rational approach to maximize renoprotection in patients with CKD. Drugs are also being developed that may reduce renal disease progression by targeting the mechanisms downstream of proteinuria. In this line, pirfenidone, a TGF-beta inhibitor, reduced renal function loss in small studies with patients with FSGS or diabetes; however, the high dropout rate raised serious concerns about the safety of this compound, particularly in patients with diabetes. Traditional recommendations regarding diet in the setting of CKD have focused on limiting protein and dietary acid intake. Experimental evidence suggests that protein intake increases intraglomerular pressure and causes hyperfiltration in the kidneys. In individuals with obesity, weight loss may reduce the risk of CKD progression and improve renal function. Exercise benefits patients with chronic kidney disease (CKD). Several small randomized trials have reported that exercise training programs in patients with moderate-to-severe CKD are safe, feasible, and effective in improving physical activity levels, cardiorespiratory fitness, and QoL. Whether these interventions also slow CKD progression remains to be determined, as many of these studies were underpowered to determine this outcome. Renal replacement therapy is necessary when kidney function continues to decline despite treatment. It is used in end-stage renal disease to replace the lost kidney function. It includes hemodialysis, peritoneal dialysis, and kidney transplantation, which remove waste products, correct fluid and electrolyte imbalances, and improve the survival and quality of life of patients with advanced chronic kidney disease.

**Recent Advancement in CKD Management**

The mainstay of chronic kidney disease (CKD) management involves treating the underlying cause whenever it is identified and controlling risk factors, such as albuminuria, which contribute to disease progression and the development of CKD-related complications.

**Reducing the Risk of Cardiovascular Disease:** The prevalence of cardiovascular disease is a major concern in patients with chronic kidney disease (CKD), as it is significantly higher in this population than in individuals without CKD. CKD contributes to cardiovascular risk through factors such as hypertension, dyslipidemia, inflammation and vascular calcification. Early CKD detection, strict blood pressure control, diabetes management, lifestyle modification, and appropriate pharmacotherapy are essential for lowering cardiovascular morbidity and mortality.

**Hypertension control:** Many guidelines provide algorithms detailing the agents that should be used to treat hypertension in patients with CKD. The presence and severity of albuminuria should be evaluated. Blockade of the renin-angiotensin-aldosterone system with either an angiotensin-converting enzyme inhibitor (ACE-I) or an angiotensin II receptor blocker (ARB) is recommended for adults with diabetes.

**Glycemic control:** Glycemic control is an important component of comprehensive care for patients with diabetes and CKD. First, glycemic control may delay the progression of CKD. Second, dose adjustment of oral hypoglycemic agents may be necessary. Third, the use of specific medication classes, such as SGLT-2 inhibitors, in patients with severely increased albuminuria should be considered.

**Nephrotoxins:** All patients with CKD should be counseled to avoid nephrotoxins. The routine administration of NSAIDs in CKD is not recommended, especially in individuals receiving ACE-I or ARB therapy.

**Drug Dosing:** Adjustments in drug dosing are frequently required in patients with CKD. Common medications that require dose reduction include

antibiotics, direct oral anticoagulants, gabapentin, pregabalin, oral hypoglycemic agents, insulin, chemotherapeutic agents and opiates. In general, the use of medications with a low likelihood of benefit should be minimized because patients with CKD are at a high risk of adverse drug events.

**Dietary management:** Individuals with kidney disease are usually advised to change their diets. A low-protein diet (0.81 g/kg/day) is often recommended to help slow down the buildup of waste in the body and limit nausea and vomiting that can accompany chronic renal failure.

**Specific Classes of Therapy**

**Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers:** Antihypertensive agents that act by inhibiting the renin-angiotensin-aldosterone system (RAASi) are particularly relevant in patients with CKD. The renin-angiotensin system (RAS) is involved in the progression of renal disease, and it was hypothesized that continued disease progression could be prevented by a more complete RAS blockade. To investigate this, several trials have investigated the combination of ACEi and ARB have been performed.

**Renin Inhibition:** An alternative approach to RAS blockade is to inhibit the first step in the renin-angiotensin-aldosterone cascade, renin-mediated cleavage of angiotensinogen to angiotensin-1. The effect of the renin inhibitor aliskerin on renal outcomes was tested in patients with diabetic nephropathy.

**SGLT-2 inhibitors:** One of the most significant advancements in CKD management over the past decade is the discovery that SGLT-2 inhibitors have robust protective effects on the heart and kidneys in patients with and without diabetes. Recent trials have demonstrated an approximate 30% reduction in the risk of diverse kidney outcomes among individuals with baseline eGFR values as low as 20 ml/min/1.73 $m^2$.

**Mineralocorticoid receptor antagonists:** Several MRAs are available and can be useful adjuncts to RAASi therapy, particularly in patients with albuminuria and/or diabetes mellitus. Two common steroidal non-selective MRAs, spironolactone and eplerenone, lower albuminuria levels

**GLP-1 receptor agonists:** GLP-1 RA have also been shown to improve kidney outcomes in individuals with type 2 diabetes, albeit in trials designed for primary cardiac outcomes. The reduction in the risk of kidney outcomes, including albuminuria, ranged from 15% to 36%.

**Endothelin Receptor Antagonists:** Preclinical studies in animal models of kidney disease have suggested that selective blockade of the endothelin A (ETA) receptor is associated with renal protection when used in addition to existing therapies, such as RAS interventions.

**Monitoring of Established CKD and Follow Up**

Once CKD is established, the KDIGO guidelines recommend monitoring eGFR and albuminuria at least once a year. For patients at high risk, these measures should be monitored at least twice per year, and patients at very high risk should be monitored at least three times per year. Patients with moderate-to-severe CKD are at an increased risk of developing electrolyte abnormalities, mineral and bone disorders, and anemia. The screening and frequency of assessment for laboratory abnormalities are dictated by the stage of CKD and include the measurement of complete blood count and lipid levels.

**Public Health**

Any health problem can be called a public health issue if it fulfills the following four essential criteria:

- If the risk is higher, then that is, its number has increased significantly in a short time or has a higher chance of increasing.
- If it affects a larger population unevenly, it mainly affects poor and vulnerable populations.
- If there is evidence that proper preventive steps can significantly decrease the risk of the disease.
- These preventive steps must be implemented properly.

CKD fulfills all four criteria for being considered a public health issue. Starting with the first criterion, more than 850 million patients are globally affected by CKD, and this number is expected to rise in the near future. CKD is associated with the risk of premature morbidity and mortality and a high risk of Cardiovascular Diseases (CVD). CKD is also associated with a poor quality of life and increased dependency on healthcare. CKD also meets the second criterion. According to multiple international data, CKD poses a global public-health challenge. As per the study of the Global Burden of Disease (GBD), the incidence of CKD has increased between 1990 and 2017 by a much higher rate of 30%. CKD also meets the third and fourth public health criteria. Even though it is a high-risk disease, there is some positive news regarding it: we have sufficient strategies and resources to prevent its incidence, delay its progression, or prevent its progression through early diagnosis. According to the Global Kidney Health Atlas data, the strategies of different nations to address CKD are positively proportional to their income levels.

| Income Level Of country | Availability of Strategy |
|---|---|
| Lower Income Country (LIC) | 11% |
| Lower | 23% |
| Upper | 22% |
| Higher Income Country (HIC) | 33% |

Some countries have formed policies regarding CKD and run awareness programs, but the majority of the population is still not aware of CKD, its effects on the population, how it leads to other diseases, and its financial burden. CKD is most prevalent in individuals with other chronic diseases/disorders. Therefore, the government and policymakers should formulate policies that not only correlate with policies regarding other chronic diseases but also ensure better healthcare facilities for treating CKD.

## EMERGING TREATMENTS AND FUTURE PROSPECTS

In recent years, several new therapeutic methods have been developed for the management of CKD. Some clinical trials are in phases 3 and 4 to assess new CKD treatments. These include the use of dapagliflozin to effectively slow CKD progression in the early stages. Recently, this drug was approved by the United States Food and Drug Administration (US FDA) for the treatment of CKD. Another drug, finerenone, a non-steroidal oral mineralocorticoid receptor antagonist (MRA), has shown a decreased chance of CKD progression and cardiovascular episodes in diabetic kidney disease (DKD). It is under investigation for approval by the European Medicines Agency (EMA) and the United States Food and Drug Administration (US FDA). There are many other therapies which are under investigation like Obinutuzumab, Zaubrutinab in membranous nephropathy; Vaclosporin Anifrolumab, Inanalumab, Secukinumab and Obinutuzumab in lupus nephritis; Sparsentan in focal segmental glomerulosclerosis and pegcetacoplan in immune complex glomerulonephritis. Many other therapies are under investigation. In addition to these investigational and clinical studies, an integrated approach is needed to decrease the risk of CKD progression in patients with diabetes. There will be benefits from the integration of research into Clinical Practice, using electronic health records, applying personalized medicine approaches, and incorporating artificial intelligence (AI) for data analysis. To accelerate the development of precision drug delivery and targeted therapy, better cooperation among nephrologists, pharmacology experts, data scientists, biostatisticians, engineers, and AI experts is required. Patients should also be encouraged to actively participate in treatment and research programs aided by mobile applications, websites, and awareness programs. It is essential to develop an interdisciplinary and forward-looking workforce for investing and collaborating from laboratories to clinical setups to improve care for everyone with kidney disease or at risk of developing kidney disease. Telemedicine can be promoted, which helps in remote observation and consultations, improving care, especially in rural and remote areas of the country. Personalized treatment can be promoted, which works on individual patient preferences and conditions. To ensure that all information reaches the people in need,

public health agencies, healthcare providers and local community institutions should collaborate and convey the information

## CONCLUSION

CKD is growing globally, affecting 8% to 16% of the population and is a major cause of death in the United States. Apart from lifestyle changes and associated CKD risk factors, it is also essential to identify chronic early and start treatment for complications, which include anemia, metabolic acidosis, and hyperkalemia, and reduce the morbidity and mortality in populations affected by CKD. To decrease the global burden of CKD, clinicians are important in the diagnosis, staging, and proper recommendation of CKD. Recent advancements and novel therapeutics may improve treatment and reduce CKD progression.

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