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Review Article

CHRONIC KIDNEY DISEASE: GLOBAL BURDEN, RISK FACTORS, AND THERAPEAUTIC PERSPECTIVES

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ABSTRACT

Chronic kidney disease (CKD) is a progressive and irreversible condition characterized by a gradual loss of kidney function over time and is recognized as a major global public health problem. CKD is defined by abnormalities in kidney structure or function persisting for more than three months and is commonly identified by a reduced glomerular filtration rate (GFR) or the presence of markers of kidney damage such as albuminuria. The global prevalence of CKD has increased significantly in recent decades and currently affects approximately 10–13% of the world's population, contributing substantially to morbidity, mortality, and healthcare costs.

The major causes of CKD include diabetes mellitus, hypertension, glomerulonephritis, and genetic or hereditary kidney disorders. Additional risk factors such as obesity, aging, cardiovascular diseases, and exposure to nephrotoxic agents further contribute to the growing burden of the disease. The underlying pathophysiological mechanisms involve progressive nephron loss, glomerular hyperfiltration, activation of the renin–angiotensin–aldosterone system, oxidative stress, inflammation, and renal fibrosis, which collectively lead to a gradual decline in renal function and the development of end-stage renal disease (ESRD).

Current management strategies focus primarily on slowing disease progression, controlling underlying causes, and preventing complications. These include lifestyle modifications, strict glycaemic and blood pressure control, use of renin–angiotensin system inhibitors, sodium–glucose cotransporter-2 (SGLT2) inhibitors, and other supportive pharmacological therapies. In advanced stages, renal replacement therapies such as dialysis or kidney transplantation are required to sustain life.

Despite advances in treatment, CKD continues to impose a significant health and economic burden worldwide. Future perspectives emphasize the importance of early detection, improved screening programs for high-risk populations, development of novel therapeutic targets, and implementation of effective preventive strategies. Strengthening multidisciplinary care and public health interventions will be crucial to reduce the global impact of CKD and improve patient outcomes.

KEYWORDS: Chronic Kidney Disease, GFR, KDGIO, DM, HTN, Anemia.

Introduction

Chronic kidney disease (CKD) is a progressive and irreversible disorder characterized by abnormalities in kidney structure or function that persist for more than three months and have important health implications. The kidneys play a crucial role in maintaining homeostasis by regulating fluid balance, electrolyte levels, blood pressure, and the removal of metabolic waste products. When kidney function declines gradually over time, these essential physiological processes become impaired, ultimately leading to serious systemic complications[1].

The diagnosis of CKD is based on specific clinical criteria established by international guidelines. CKD is defined by a reduced glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for a duration exceeding three months, or by the presence of markers of kidney damage[2]. These markers may include albuminuria, abnormalities in urine sediment, structural abnormalities detected through imaging, or histological changes identified through renal biopsy[3]. Based on the level of GFR and the degree of albuminuria, CKD is classified into different stages, which help guide clinical management and risk assessment[4].

CKD has emerged as a significant global health burden affecting millions of individuals worldwide. Recent estimates suggest that approximately 10–13% of the global population is affected by some form of CKD, making it one of the leading causes of morbidity and mortality. The rising prevalence of CKD is closely associated with the increasing incidence of diabetes mellitus, hypertension, obesity, and aging populations. Additionally, lifestyle changes and environmental factors have further contributed to the growing number of individuals at risk of developing kidney disease[5].

The impact of CKD extends beyond its clinical consequences and places a substantial economic burden on healthcare systems. Patients with CKD often require long-term medical management, frequent monitoring, and treatment for multiple complications such as anaemia, cardiovascular disease, and metabolic bone disorders. In advanced stages, renal replacement therapies including dialysis and kidney transplantation become necessary, which are costly and may not be accessible to all patients, particularly in low- and middle-income countries[6].

Given the progressive nature of CKD and the limited treatment options available in later stages, early detection and timely intervention are essential to prevent or delay disease progression. Screening programs targeting high-risk populations, improved awareness among healthcare providers, and the implementation of preventive strategies can significantly reduce the burden of CKD. Therefore, understanding the epidemiology, underlying mechanisms, and management approaches of CKD is crucial for improving patient outcomes and reducing its global health impact[7].

Epidemiology

Chronic kidney disease (CKD) has emerged as a major global health concern due to its increasing prevalence and significant impact on morbidity and mortality. Recent epidemiological studies estimate that CKD affects approximately 10–13% of the global population, making it one of the most common non-communicable diseases worldwide. The growing burden of CKD is largely attributed to the rising prevalence of major risk factors such as diabetes mellitus, hypertension, obesity, and aging populations. In addition, lifestyle changes, urbanization, and environmental factors have contributed to the increasing incidence of kidney disease in both developed and developing countries[8].

In India, CKD has become an important public health problem with a steadily rising prevalence. Population-based studies suggest that the prevalence of CKD in India ranges from 8% to 17%, depending on the study population and diagnostic criteria used. The increasing incidence of diabetes and hypertension, which are the leading causes of CKD, has significantly contributed to this growing burden. Limited awareness, delayed diagnosis, and restricted access to specialized healthcare services in rural areas further exacerbate the problem, leading to many patients presenting at advanced stages of the disease[9].

The distribution of CKD varies across different age groups and genders. The prevalence of CKD generally increases with age due to the natural decline in kidney function and the higher occurrence of associated comorbidities such as cardiovascular disease and metabolic disorders among older adults. Although CKD affects both men and women, several studies have reported a slightly higher prevalence in women, whereas men often experience faster disease progression and a higher risk of developing end-stage renal disease (ESRD)[10].

CKD is also recognized as a leading cause of morbidity and mortality worldwide. It significantly increases the risk of cardiovascular complications, which remain the primary cause of death among CKD patients. As kidney function declines, patients may develop serious complications such as anaemia, metabolic acidosis, electrolyte imbalances, and bone mineral disorders. Furthermore, advanced CKD frequently requires renal replacement therapies, including dialysis or kidney transplantation, which impose substantial clinical, social, and economic burdens on patients, families, and healthcare systems. Consequently, the growing epidemiological burden of CKD highlights the urgent need for effective prevention strategies, early diagnosis, and improved management approaches[11].

Etiology and Risk Factors

Chronic kidney disease (CKD) develops as a result of a variety of underlying conditions that cause gradual and irreversible damage to the kidneys. Several diseases and

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lifestyle-related factors contribute to the development and progression of CKD. Identifying these causes and risk factors is essential for early diagnosis, prevention, and effective management of the disease[12].

Among the major causes, diabetes mellitus is considered the leading contributor to CKD worldwide. Persistent hyperglycaemia in diabetic patients leads to structural and functional changes in the kidneys, including glomerular hyperfiltration, thickening of the glomerular basement membrane, and progressive nephron damage, ultimately resulting in diabetic nephropathy. Hypertension is another major cause, as prolonged high blood pressure damages renal blood vessels and reduces the kidneys' ability to effectively filter waste products from the blood[13].

Glomerulonephritis, a group of inflammatory disorders affecting the glomeruli, also contributes significantly to CKD. These conditions may arise due to immune-mediated mechanisms, infections, or systemic diseases, leading to progressive scarring and loss of kidney function. In addition, polycystic kidney disease (PKD), a hereditary disorder characterized by the formation of multiple fluid-filled cysts in the kidneys, gradually enlarges the kidneys and impairs their normal function, eventually leading to chronic kidney failure[14].

In addition to these primary causes, several other risk factors increase the likelihood of developing CKD. Obesity is associated with metabolic abnormalities, insulin resistance, and increased glomerular filtration pressure, which can accelerate kidney damage. Smoking has been shown to worsen kidney function by promoting oxidative stress, endothelial dysfunction, and vascular injury. Similarly, cardiovascular diseases are closely linked with CKD, as both conditions share common risk factors such as hypertension, diabetes, and dyslipidaemia[15].

Exposure to nephrotoxic drugs, including certain nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics, and chemotherapeutic agents, can also contribute to kidney injury when used for prolonged periods or in high doses. Furthermore, genetic predisposition plays an important role in some individuals, making them more susceptible to kidney diseases due to inherited mutations or familial conditions. Aging is another important risk factor, as kidney function naturally declines with age due to progressive loss of nephrons and reduced renal blood flow[16].

Overall, CKD results from a complex interaction between underlying diseases, lifestyle factors, genetic susceptibility, and environmental influences. Understanding these etiological factors is crucial for implementing preventive strategies and reducing the global burden of chronic kidney disease.

Pathophysiology

The pathophysiology of chronic kidney disease (CKD) involves a complex series of structural and functional changes within the kidneys that progressively impair their ability to filter blood and maintain homeostasis. CKD typically begins with the loss of functional nephrons, the basic filtration units of the kidney. As nephron loss occurs due to underlying diseases or injury, the remaining nephrons undergo compensatory hyperfiltration in an attempt to maintain overall kidney function. Although this mechanism initially helps sustain glomerular filtration, prolonged hyperfiltration increases intraglomerular pressure and eventually leads to further nephron injury and progressive kidney damage[17].

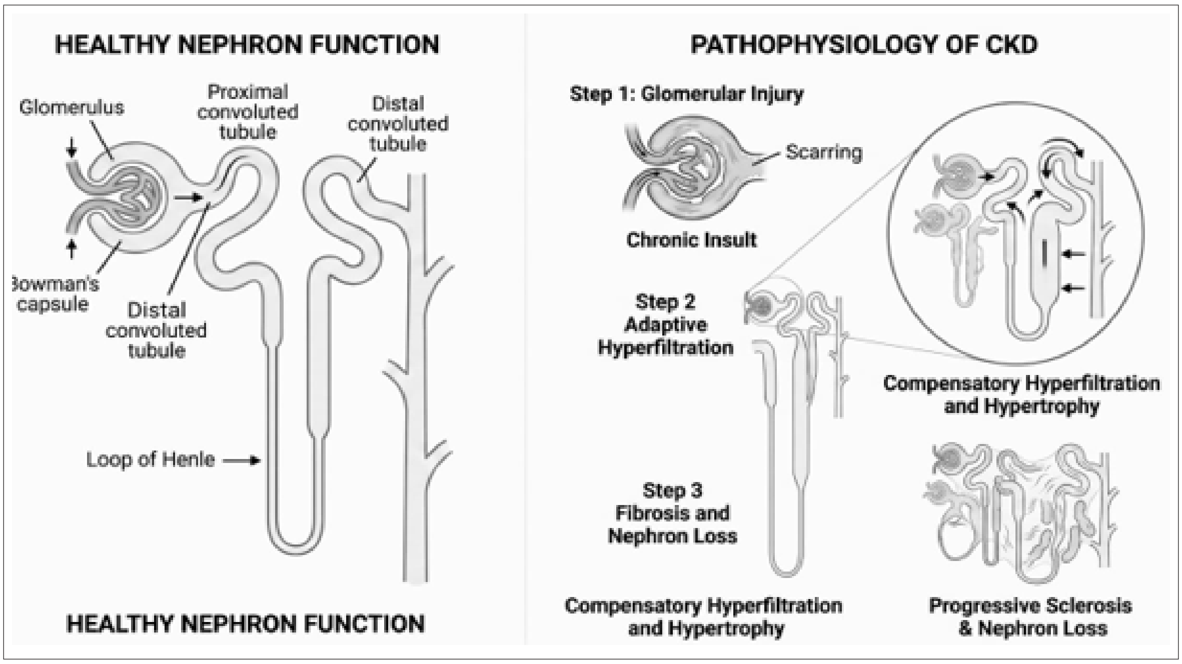

Figure 1: Pathophysiology of CKD

A key mechanism involved in CKD progression is the activation of the renin–angiotensin–aldosterone system (RAAS). Reduced renal perfusion and nephron damage stimulate RAAS activity, leading to vasoconstriction, sodium and water retention, and increased blood pressure. Elevated levels of angiotensin II promote inflammation, cellular proliferation, and fibrosis within renal tissues, thereby accelerating kidney damage[18].

As CKD advances, structural changes such as glomerulosclerosis occur, which involves scarring and hardening of the glomeruli. This process reduces the filtering capacity of the kidneys and contributes to a gradual decline in glomerular filtration rate (GFR). In addition to glomerular injury, damage also affects the renal tubules and surrounding interstitial tissue, resulting in tubulointerstitial fibrosis, characterized by tubular atrophy, extracellular matrix accumulation, and chronic inflammation[19].

Oxidative stress and inflammatory processes further contribute to CKD progression. The excessive production of reactive oxygen species (ROS) damages cellular components, including proteins, lipids, and DNA, while inflammatory mediators promote tissue injury and fibrosis. These processes collectively worsen renal dysfunction and accelerate disease progression[20].

Another important factor in CKD pathogenesis is endothelial dysfunction, which impairs the normal function of blood vessels within the kidneys. Damage to the endothelial lining reduces nitric oxide availability, promotes vascular stiffness, and disrupts normal renal blood flow. This contributes to further ischemic injury and deterioration of kidney function[21].

Overall, the pathophysiology of CKD involves a vicious cycle of nephron loss, compensatory hyperfiltration, hormonal activation, oxidative stress, inflammation, and progressive structural damage, ultimately leading to irreversible loss of kidney function and the development of end-stage renal disease (ESRD).

Classification and Staging

Chronic kidney disease (CKD) is commonly classified and staged based on glomerular filtration rate (GFR) and albuminuria[22].

Classification Based on GFR (G category)

Table 1: GFR reflects kidney function and is divided into five stages

GFR Category	GFR (mL/min/1.73 m²)	Description
G1	≥ 90	Normal or high kidney function
G2	60–89	Mildly decreased kidney function
G3a	45–59	Mildly to moderately decreased kidney function
G3b	30–44	Moderately to severely decreased kidney function
G4	15–29	Severely decreased kidney function
G5	<15 or dialysis	Kidney failure

Note: G3 is often subdivided into G3a and G3b to better reflect risk progression.

Classification Based on Albuminuria (A category)

Table 2: Albuminuria is an early marker of kidney damage and is classified into three categories

Albuminuria Category	Albumin-to-Creatinine Ratio (ACR, mg/g)	Description
A1	<30	Normal to mildly increased
A2	30–300	Moderately increased
A3	>300	Severely increased

Clinical Manifestations

The clinical presentation of chronic kidney disease (CKD) varies according to the stage of disease. Early recognition is often difficult because initial kidney damage is usually asymptomatic, whereas late-stage CKD presents with multiple systemic manifestations.

Early Stage

- Asymptomatic: Patients in G1–G2 stages with preserved GFR often show no overt symptoms. Kidney damage may be detected only through laboratory tests (e.g., mildly reduced GFR, microalbuminuria, or abnormal imaging).
- Subtle signs: Occasionally, mild hypertension or slight fatigue may be the only indicators[23].

Late Stage

As CKD progresses to G3–G5, the decline in kidney function

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leads to accumulation of toxins and fluid imbalances, resulting in the following clinical manifestations:

1. Edema: Fluid retention occurs due to impaired sodium and water excretion, often presenting as peripheral edema, periorbital puffiness, or ascites.
2. Fatigue: Uremic toxins and anaemia contribute to generalized weakness and reduced exercise tolerance.
3. Uraemia: Accumulation of nitrogenous wastes leads to nausea, vomiting, anorexia, and altered mental status. Severe cases may develop uremic pericarditis or encephalopathy.
4. Anaemia: Decreased erythropoietin production by the diseased kidneys results in normocytic, normochromic anaemia, causing pallor and dyspnoea on exertion.
5. Bone and Mineral Disorders: CKD–mineral and bone disorder (CKD–MBD) arises from impaired vitamin D metabolism, hyperphosphatemia, and secondary hyperparathyroidism, leading to bone pain, fractures, and skeletal deformities.
6. Electrolyte Imbalance: Disturbances such as hyperkalaemia, metabolic acidosis, hypercalcemia, and hyperphosphatemia are common, potentially causing cardiac arrhythmias and neuromuscular symptoms.

Early detection through screening and lab investigations is essential, as many late-stage complications are preventable or manageable with timely intervention[24].

Diagnosis

The diagnosis of chronic kidney disease (CKD) involves a combination of laboratory tests, imaging, and occasionally histopathology, aimed at assessing kidney function, detecting structural abnormalities, and identifying underlying causes. Early diagnosis is crucial to slow disease progression and prevent complications[25].

eGFR Calculation

Estimated glomerular filtration rate (eGFR) is the most widely used measure of kidney function. It is calculated using serum creatinine along with demographic variables such as age, sex, and race. eGFR allows staging of CKD according to the KDIGO guidelines, enabling risk stratification[26].

Urinary Albumin-to-Creatinine Ratio (UACR)

The UACR is a sensitive marker for kidney damage, especially in the early stages. Microalbuminuria (30–300 mg/g) indicates moderate kidney injury. It is often preferred over 24-hour urine collection due to convenience and accuracy[27].

Serum Creatinine

Serum creatinine reflects renal excretory function, though it may remain normal in early CKD. This happens due to excessive filtration. Rising levels indicate declining kidney function, making it a simple, widely available diagnostic marker[28].

Imaging

Renal ultrasound is the first-line imaging modality to assess kidney size, structure, and obstruction. Other imaging techniques (CT or MRI) may be used in selected cases to evaluate anomalies, cysts, or masses[29].

Renal Biopsy

Indicated in atypical presentations or when the cause of CKD is uncertain. Provides histopathological diagnosis, helping guide specific therapy, especially in glomerular diseases[30].

Emerging Biomarkers

Novel biomarkers are being explored for early detection and prognosis:

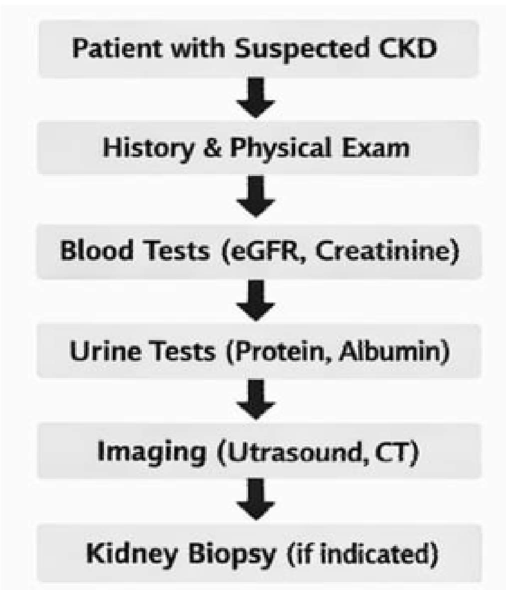

Figure 2: CKD diagnosis flowchart

- NGAL (Neutrophil Gelatinase-Associated Lipocalin) – rises early in acute and chronic kidney injury.
- KIM-1 (Kidney Injury Molecule-1) – indicates tubular injury.
- Cystatin-C – independent of muscle mass, more sensitive than creatinine for GFR estimation.

A combination of functional tests, structural assessment, and biomarkers ensures accurate diagnosis, early intervention, and better risk prediction in CKD[31].

Complications

Chronic kidney disease (CKD) is associated with multiple systemic complications that increase morbidity and mortality. These arise due to the progressive decline in kidney function, accumulation of uremic toxins, electrolyte imbalances, and hormonal dysregulation[32].

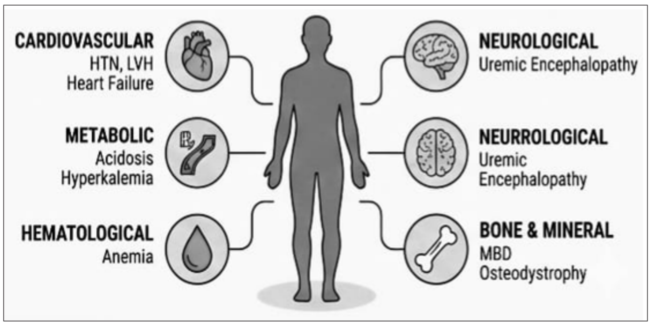

Figure 3: Complications of CKD

Cardiovascular Disease

Heart failure, stroke, coronary artery disease, hypertension, and other cardiovascular events are all significantly increased by chronic kidney disease (CKD). Mechanisms include accelerated atherosclerosis, vascular calcification, and chronic inflammation[33].

Anaemia

Results primarily from reduced erythropoietin production by the kidneys. Leads to fatigue, pallor, and reduced exercise tolerance, contributing to cardiovascular strain[34].

CKD–Mineral and Bone Disorder (CKD–MBD)

Impaired calcium, phosphate, and vitamin D metabolism leads to secondary hyperparathyroidism. Clinical consequences include bone pain, fractures, skeletal deformities, and vascular calcifications[35].

Metabolic Acidosis

Reduced acid excretion in CKD causes chronic metabolic acidosis, resulting in muscle wasting, bone demineralization, and impaired growth in children[36].

Hyperkalaemia

Impaired renal potassium excretion can lead to elevated serum potassium, increasing the risk of life-threatening cardiac arrhythmias[37].

Fluid Overload

Due to sodium and water retention, patients may present with peripheral oedema, pulmonary congestion, and hypertension[38].

Uraemia

Nitrogenous waste accumulation causes neuropathy, nausea, vomiting, anorexia, pruritus, and cognitive decline. Severe uraemia may necessitate renal replacement therapy[39]. Early identification and management of these complications are essential to improve quality of life, reduce hospitalization, and delay progression to end-stage renal disease.

Management

The management of chronic kidney disease (CKD) aims to slow disease progression, control complications, and improve patient quality of life. Treatment strategies involve non-pharmacological interventions and pharmacological therapy, often used in combination[40].

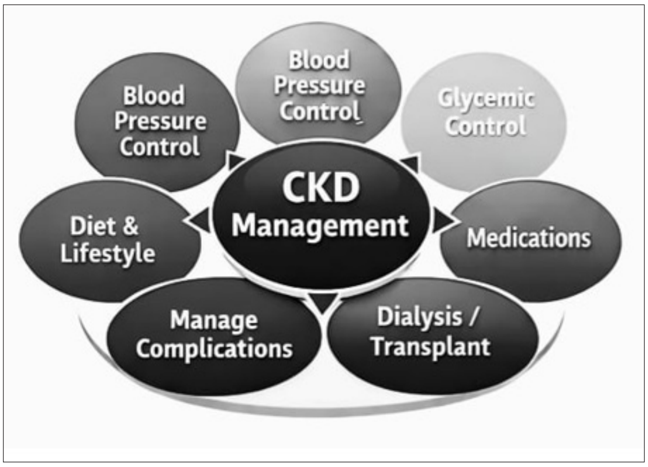

Figure 5: CKD Management

Non-Pharmacological Management

Lifestyle modifications play a crucial role in CKD management:

- Dietary Protein Restriction: Moderate reduction of protein intake helps decrease glomerular hyperfiltration and uremic toxin accumulation.
- Salt Restriction: Limiting sodium intake helps control blood pressure and reduce fluid retention.
- Weight Management: Maintaining a healthy body weight reduces cardiovascular risk and slows CKD progression.
- Smoking Cessation: Smoking accelerates vascular damage and CKD progression; cessation is strongly recommended.
- Physical Activity: Regular exercise improves cardiovascular health, insulin sensitivity, and overall well-being.

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Pharmacological Management

Pharmacological interventions are tailored based on CKD stage, comorbidities, and complications:

- RAAS Inhibitors (ACEIs/ARBs): Reduce proteinuria, control blood pressure, and slow kidney disease progression.
- SGLT2 Inhibitors: Particularly in diabetics, these drugs reduce hyperfiltration, lower glucose, and provide cardiovascular protection.
- GLP-1 Receptor Agonists (in diabetics): Improve glycaemic control and may offer Reno protective effects.
- Diuretics: Useful for controlling fluid overload and hypertension, especially in advanced CKD.
- Statins: Lower LDL cholesterol to reduce cardiovascular risk, a major cause of morbidity in CKD.
- Erythropoiesis-Stimulating Agents: Correct anaemia resulting from reduced erythropoietin production.
- Phosphate Binders: Manage hyperphosphatemia to prevent CKD–MBD.
- Vitamin D Analogues: Address secondary hyperparathyroidism and improve bone health.

A multidisciplinary approach, combining lifestyle modifications and targeted pharmacotherapy, is essential for optimal CKD care. Early intervention can delay progression to end-stage renal disease (ESRD) and improve long-term outcomes[41].

Renal Replacement Therapy

Renal replacement therapy (RRT) is indicated in patients with end-stage renal disease (ESRD) or severe CKD (G5) when kidney function is insufficient to maintain homeostasis. RRT aims to replace lost renal function, remove toxins, correct fluid and electrolyte imbalances, and improve quality of life.

Haemodialysis

Involves extracorporeal blood filtration using a dialyzer to remove waste products and excess fluids. Typically performed 3 times per week for 3–5 hours per session. • Requires vascular access (e.g., arteriovenous fistula, graft, or central venous catheter). Effective in rapid toxin removal, but associated with hemodynamic fluctuations and vascular complications[42].

Peritoneal Dialysis

Utilizes the peritoneal membrane as a natural semipermeable membrane for solute and fluid exchange. Can be performed at home either manually (CAPD) or via automated cyclers (APD). Advantages include flexibility, independence, and preservation of residual kidney function, but carries risk of peritonitis and catheter-related infections[43].

Kidney Transplantation

Considered the definitive treatment for ESRD. Offers better long-term survival, improved quality of life, and freedom from dialysis. Requires compatible donor (living or deceased) and lifelong immunosuppressive therapy to prevent graft rejection. Complications may include rejection, infection, and medication-related side effects. Selection of RRT modality depends on patient age, comorbidities, lifestyle, availability of resources, and personal preference. Early planning for RRT in CKD patients improves treatment outcomes and reduces morbidity[44].

Role of Pharmacist in CKD

Pharmacists play a critical role in the management of chronic kidney disease (CKD), contributing to both clinical outcomes and patient quality of life. Their expertise ensures safe and effective use of medications while preventing further renal damage.

Dose Adjustment in Renal Impairment

Pharmacists review medications and adjust doses based on eGFR or creatinine clearance to prevent drug accumulation and toxicity. Special attention is given to renally excreted drugs such as aminoglycosides, digoxin, and certain antivirals.

Prevention of Nephrotoxicity

Identify and modify or avoid nephrotoxic agents, including NSAIDs, contrast agents, and certain antibiotics. Collaborate with physicians to select safer alternatives for patients with reduced renal function.

Patient Counselling

Educate patients on disease progression, lifestyle modifications, dietary restrictions, and medication use. Provide guidance on recognizing early warning signs of complications such as fluid overload, hyperkalaemia, or uremic symptoms.

Medication Adherence

Support adherence through simplified dosing schedules, reminder systems, and patient education. Emphasize the importance of consistent medication use to prevent CKD progression and complications.

Monitoring Drug Interactions

Evaluate polypharmacy, especially in CKD patients with comorbidities like diabetes, hypertension, or cardiovascular disease. Prevent harmful drug–drug interactions and adjust therapy accordingly.

Participation in Multidisciplinary Care

Collaborate with nephrologists, dietitians, nurses, and other healthcare professionals to provide comprehensive CKD care. Contribute to treatment planning, monitoring therapeutic outcomes, and optimizing renal-protective strategies. By actively participating in CKD management, pharmacists enhance patient safety, improve therapeutic outcomes, and support the overall effectiveness of multidisciplinary care teams [45].

Novel Therapies and Research Advances

Recent years have witnessed significant progress in therapeutic strategies and research aimed at slowing CKD progression, reducing complications, and improving patient outcomes. These novel approaches target specific pathophysiological pathways and hold promise for personalized management of CKD.

Finerenone

A non-steroidal mineralocorticoid receptor antagonist that reduces cardiovascular and renal events in CKD patients, particularly those with diabetes. Demonstrates lower risk of hyperkalaemia compared to traditional mineralocorticoid receptor blockers.

Endothelin Receptor Antagonists

Target the endothelin pathway, which is implicated in vasoconstriction, inflammation, and fibrosis in CKD. These agents have shown promise in reducing proteinuria and slowing kidney function decline.

Anti-fibrotic Agents

Aim to limit renal fibrosis, a key mechanism of CKD progression. Experimental drugs targeting TGF-β signalling and other profibrotic pathways are under investigation to preserve nephron integrity.

Stem Cell Therapy

Mesenchymal stem cells (MSCs) and other regenerative approaches are being explored to repair damaged renal tissue and promote regeneration. Preclinical studies demonstrate improved renal function and reduced inflammation, though clinical application is still in early stages.

Precision Medicine

Involves tailoring therapy based on genetic, molecular, and phenotypic patient characteristics. Enables personalized risk assessment, optimized drug selection, and targeted interventions, potentially enhancing treatment efficacy and minimizing adverse effects.

These novel therapies and research advances reflect a shift toward mechanism-based, patient-specific management in CKD (189). While some are already in clinical use, ongoing studies continue to explore their safety, efficacy, and long-term outcomes[46].

Prevention Strategies

Prevention of chronic kidney disease (CKD) focuses on early detection, risk factor modification, and minimizing kidney injury. Effective prevention not only slows disease progression but also reduces morbidity and healthcare burden.

Early Screening in High-Risk Patients

High-risk populations include individuals with diabetes, hypertension, cardiovascular disease, family history of CKD, and elderly patients. Early screening using eGFR and urinary albumin-to-creatinine ratio (UACR) allows timely intervention before significant kidney damage occurs.

Glycaemic and Blood Pressure Control

Optimal glycaemic control in diabetic patients and blood pressure management are key strategies to prevent CKD onset and progression. Targeted therapy with RAAS inhibitors or SGLT2 inhibitors offers renal protection in high-risk patients.

Lifestyle Modification

Maintaining a healthy weight, regular physical activity, balanced diet, and smoking cessation reduces the risk of CKD. Limiting salt intake and moderating protein consumption can further protect renal function.

Avoidance of Nephrotoxic Drugs

Reducing exposure to NSAIDs, certain antibiotics, radiographic contrast agents, and other nephrotoxic medications is essential. Pharmacists and healthcare providers play a critical role in medication review and patient counselling to prevent iatrogenic kidney injury.

Implementation of these primary and secondary prevention strategies can significantly reduce the incidence and progression of CKD, improving both patient outcomes and long-term quality of life[47].

Future Perspectives

The management of chronic kidney disease (CKD) is evolving rapidly, with emerging strategies aimed at early detection, individualized treatment, and improved patient outcomes. Future research focuses on leveraging advanced technologies and precision medicine to transform CKD care.

Biomarker-Based Early Diagnosis

New biomarkers like cystatin-C, KIM-1, and NGAL may make it possible to identify kidney damage earlier before a major reduction in function takes place. Early identification allows timely intervention, potentially preventing progression to advanced CKD or end-stage renal disease.

Artificial Intelligence in CKD Prediction

Artificial intelligence (AI) and machine learning algorithms are being developed to predict CKD onset, progression, and complication risk based on clinical, laboratory, and imaging data. These tools can support personalized monitoring and decision-making, optimizing treatment strategies for individual patients.

Personalized Therapy

Future CKD management will increasingly focus on precision medicine, tailoring interventions to a patient's genetic, molecular, and phenotypic profile. Personalized therapy may include targeted pharmacological treatments,

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lifestyle recommendations, and regenerative approaches, improve efficacy while minimize adverse effects.

Advances in biomarkers, AI, and personalized medicine hold great promise for redefining CKD management, shifting the paradigm from reactive care to preventive and individualized interventions[48].

Conclusion

Chronic kidney disease (CKD) is a progressive condition with significant morbidity and mortality, yet it is largely preventable and manageable with timely intervention. Early detection through screening of high-risk individuals and routine assessment of kidney function is critical for slowing disease progression and reducing complications.

A multidisciplinary approach, involving nephrologists, dietitians, nurses, and particularly pharmacists, enhances patient care through dose optimization, prevention of nephrotoxicity, patient counselling, and medication management. Emerging therapies, biomarker-based diagnostics, and personalized treatment strategies hold promise for improving outcomes and quality of life in CKD patients.

In summary, integrating early diagnosis, lifestyle modification, pharmacological therapy, and collaborative care is essential for effective CKD management and for minimizing its global health burden.

Conflict of Interest

None

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