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Review Article

Kidney-Related Diseases: An Overview of Epidemiology, Pathophysiology, Diagnosis, Management and Emerging Therapeutic Approaches

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A B S T R A C T

Kidney-related diseases constitute an increasingly important global health challenge because of their high prevalence, progressive nature, association with cardiovascular morbidity, and substantial requirement for long-term renal replacement therapy. Kidney disorders encompass a broad spectrum of conditions, including acute kidney injury, chronic kidney disease, glomerular disorders, tubulointerstitial diseases, diabetic kidney disease, hypertensive kidney disease, hereditary disorders, obstructive uropathies and kidney failure. Chronic kidney disease is particularly important because it may remain clinically silent during its early stages and is frequently identified only after substantial loss of kidney function. Recent global estimates indicate that approximately 788 million adults were living with chronic kidney disease in 2023, demonstrating the considerable and increasing worldwide burden of this condition.[1] Diabetes mellitus and hypertension remain major contributors to chronic kidney disease, while cardiovascular disease, obesity, ageing, genetic disorders, infections, autoimmune diseases, nephrotoxic exposure and previous acute kidney injury may also contribute to renal impairment.[2,3] Diagnosis relies principally on assessment of estimated glomerular filtration rate and urinary albumin excretion, supplemented by imaging, serological investigations and kidney biopsy when clinically indicated. Management has evolved substantially from conventional renin-angiotensin system blockade and risk-factor modification toward comprehensive cardiorenal protection involving sodium-glucose cotransporter-2 inhibitors, nonsteroidal mineralocorticoid receptor antagonists and individualized risk-based treatment. Recent evidence has demonstrated renal and cardiovascular benefits of dapagliflozin, empagliflozin and finerenone in appropriate populations.[4-7] This review discusses the major categories of kidney-related diseases, their pathophysiological mechanisms, diagnostic approaches, therapeutic strategies, preventive measures and emerging directions in nephrology. Greater emphasis on early detection, integrated

cardiorenal care, equitable access to treatment and implementation of evidence-based guidelines is essential for reducing the global burden of kidney disease.

Keywords: Kidney Disease, Chronic Kidney Disease, Acute Kidney Injury, Nephrology, Diabetic Kidney Disease, Glomerular Disease, Renal Failure, Dialysis, Kidney Transplantation, SGLT2 Inhibitors

Introduction

The kidneys are essential organs responsible for maintaining fluid and electrolyte homeostasis, regulating acid-base balance, eliminating metabolic waste, controlling blood pressure and participating in endocrine functions such as erythropoietin production and vitamin D metabolism. Consequently, impairment of renal structure or function can produce systemic consequences extending well beyond the urinary system. Kidney disease includes both acute and chronic disorders, with acute kidney injury characterized by an abrupt decline in kidney function and chronic kidney disease characterized by persistent abnormalities of kidney structure or function.[8] The clinical spectrum is broad and ranges from asymptomatic reduction in glomerular filtration to severe kidney failure requiring dialysis or kidney transplantation.

The global burden of kidney disease has increased substantially over recent decades. The Global Burden of Disease Study 2023 estimated that approximately 788 million adults aged 20 years or older were living with chronic kidney disease in 2023, compared with approximately 378 million in 1990.[1] India represents one of the countries with the largest absolute numbers of individuals affected by chronic kidney disease, reflecting its large population together with the increasing prevalence of diabetes, hypertension and other metabolic disorders.[1] The World Health Organization similarly recognizes kidney disease as an important contributor to morbidity, disability and premature mortality worldwide.[8]

A major challenge in kidney disease is the frequently asymptomatic nature of early renal dysfunction. Individuals may maintain relatively normal daily functioning despite significant reductions in renal reserve. This creates a substantial gap between the actual prevalence of disease and the number of people who are clinically diagnosed. Simple blood and urine investigations, particularly estimated glomerular filtration rate and urinary albumin assessment, can facilitate earlier detection.[8,9] Early identification is important because progression may be slowed through treatment of underlying causes and modification of cardiovascular and renal risk factors.

The contemporary understanding of kidney disease has also shifted from viewing renal impairment as an isolated organ-specific disorder toward recognizing the kidney as an integral component of systemic cardiovascular and metabolic disease. Chronic kidney disease is strongly associated with cardiovascular morbidity and mortality, while cardiovascular disease, diabetes and hypertension may simultaneously contribute to renal deterioration.[2,10] The present review therefore examines kidney-related diseases from an integrated clinical perspective, encompassing disease classification, epidemiology, pathophysiology, diagnosis, management, prevention and emerging therapeutic strategies.

Spectrum of Kidney-Related Diseases

Kidney-related diseases comprise heterogeneous disorders affecting different anatomical and functional components of the nephron and urinary tract. Acute kidney injury is characterized by a rapid decline in kidney function and may result from decreased renal perfusion, intrinsic renal injury or urinary tract obstruction. Common clinical settings include sepsis, severe volume depletion, major surgery, nephrotoxic exposure and critical illness. AKI may be reversible, but it is clinically important because an episode of AKI can subsequently increase the risk of chronic kidney disease and other adverse outcomes.[11] The forthcoming updated KDIGO framework further emphasizes the continuum between AKI, acute kidney disease and subsequent chronic kidney disease.[11]

Chronic kidney disease represents persistent abnormalities of kidney structure or function lasting for at least three months. It may arise from diabetes, hypertension, glomerular disease, hereditary disorders, autoimmune conditions, recurrent infection, obstruction, nephrotoxic exposure and other causes.[2,9] CKD is commonly categorized according to estimated glomerular filtration rate and albuminuria, allowing clinicians to estimate prognosis and individualize management. The 2024 KDIGO guideline places particular emphasis on a comprehensive assessment of both glomerular filtration and albuminuria, together with individualized risk assessment.[9]

Glomerular diseases constitute another major group of renal disorders. These include IgA nephropathy, membranous nephropathy, focal segmental glomerulosclerosis, minimal change disease, lupus nephritis and various forms of rapidly progressive glomerulonephritis. Such disorders may manifest with hematuria, proteinuria, nephrotic syndrome, hypertension or declining kidney function. The underlying mechanisms may include immune complex deposition, autoantibody formation, complement activation, podocyte injury or systemic autoimmune disease.

Tubulointerstitial diseases primarily involve the renal tubules and surrounding interstitial tissue. Acute and chronic

interstitial nephritis may be associated with medications, infections, autoimmune disorders or systemic diseases. Chronic tubulointerstitial injury can progressively reduce kidney function and may be accompanied by impaired concentrating ability, electrolyte abnormalities and acid-base disturbances.

Hereditary and congenital kidney disorders include autosomal dominant polycystic kidney disease, Alport syndrome, congenital anomalies of the kidney and urinary tract and other genetic abnormalities. Autosomal dominant polycystic kidney disease is characterized by progressive development of renal cysts and may eventually lead to kidney failure. Genetic evaluation is increasingly relevant in patients with suggestive family histories, atypical disease patterns or unexplained kidney failure.

Obstructive kidney diseases represent another important category. Urinary tract obstruction caused by stones, prostatic enlargement, strictures, tumors or congenital abnormalities can result in hydronephrosis and deterioration of kidney function. Prompt recognition and relief of obstruction are particularly important because prolonged obstruction can result in irreversible renal damage.

Epidemiology and Global Burden

The epidemiological importance of kidney disease is increasingly recognized because of the large population affected and its association with premature mortality. The 2023 Global Burden of Disease analysis estimated 788 million adults with CKD worldwide in 2023, with India accounting for an estimated 138 million affected adults.[1] These findings highlight the importance of CKD as a major public health problem in South Asia and other low- and middle-income settings.

The increasing burden of kidney disease is closely associated with demographic and epidemiological transitions. Population ageing, increasing prevalence of diabetes, hypertension, obesity and cardiovascular disease, together with environmental and socioeconomic determinants, have contributed to the expansion of CKD risk.[1,2] In many low- and middle-income countries, access to screening, specialist nephrology services, dialysis and transplantation remains unequal, creating an additional burden for patients and healthcare systems.[8]

The relationship between kidney disease and cardiovascular disease is particularly important. Declining kidney function and increasing albuminuria are associated with higher cardiovascular risk, and many patients with CKD are more likely to experience cardiovascular events than progress to kidney failure.[10] Consequently, contemporary kidney disease management must address both renal and cardiovascular outcomes.

Major Risk Factors and Causes

Diabetes mellitus and hypertension remain among the most important causes and risk factors for CKD. Persistent hyperglycemia produces metabolic and vascular abnormalities within the kidney, contributing to glomerular hyperfiltration, endothelial dysfunction, podocyte injury, albuminuria and progressive nephron loss. Hypertension contributes through increased intraglomerular pressure and vascular injury, while established CKD can itself promote hypertension, creating a bidirectional relationship.[2,3]

Other important risk factors include cardiovascular disease, obesity, advanced age, family history of kidney disease, inherited disorders and previous episodes of acute kidney injury.[2] Smoking and exposure to nephrotoxic medications may further increase risk. Nonsteroidal anti-inflammatory drugs, certain antimicrobials, contrast agents and other nephrotoxins may cause acute or chronic renal injury in susceptible individuals.

Autoimmune diseases, infections and systemic inflammatory disorders may produce glomerular or tubulointerstitial injury. Lupus nephritis, IgA nephropathy, vasculitis and infection-associated glomerulonephritis illustrate the diverse mechanisms by which systemic disease can affect the kidney. Genetic and structural abnormalities may also produce kidney disease independently of traditional metabolic risk factors.

Environmental and occupational exposures are increasingly recognized as potential contributors to renal disease, particularly in vulnerable populations. Heat stress, dehydration, occupational toxins and limited access to healthcare may interact with conventional risk factors. These determinants are especially relevant in populations engaged in strenuous outdoor work or living in regions with limited healthcare resources.

Pathophysiology of Kidney Disease

The pathophysiology of kidney disease varies according to the underlying disorder, but progressive nephron loss represents a common pathway in many chronic conditions. Loss of functioning nephrons increases the workload of the remaining nephrons, producing adaptive hyperfiltration. Initially compensatory, this process can eventually contribute to glomerular hypertension, structural injury and further nephron loss.

In diabetic kidney disease, metabolic abnormalities, oxidative stress, activation of inflammatory pathways and alterations in intraglomerular hemodynamics contribute to glomerular injury. Hypertension similarly produces vascular and glomerular damage through sustained pressure-related stress. Persistent proteinuria is not merely a marker of renal

injury but may also participate in tubular and interstitial damage.

Inflammation, fibrosis and maladaptive repair are central features of progressive CKD. Activation of fibroblasts, extracellular matrix deposition and disruption of normal tubular architecture may result in interstitial fibrosis and tubular atrophy. Once advanced fibrosis develops, recovery of kidney function becomes increasingly difficult.

The pathophysiology of AKI differs according to the precipitating mechanism. Prerenal injury results from inadequate renal perfusion, intrinsic injury from damage to renal tissue and postrenal injury from obstruction to urinary flow. Severe AKI can produce disturbances in potassium, acid-base balance and fluid status and may require temporary kidney replacement therapy.

Clinical Manifestations

Kidney disease may remain clinically silent for prolonged periods. Early CKD frequently produces no specific symptoms, which contributes to delayed diagnosis.[2,8] When manifestations occur, they may include fatigue, edema, altered urinary frequency, foamy urine, nausea, reduced appetite, pruritus, muscle cramps and dyspnea, particularly in advanced disease.[2]

The clinical presentation depends heavily on the underlying pathology. Glomerular disease may present with hematuria and proteinuria, whereas nephrotic syndrome is characterized by substantial protein loss, hypoalbuminemia and edema. Acute kidney injury may manifest through reduced urine output, fluid overload, electrolyte abnormalities or an increase in serum creatinine. Obstructive disease may present with colicky pain, difficulty urinating, urinary retention or hydronephrosis.

Advanced kidney failure can produce multisystem manifestations collectively associated with uremia. These may include neurological abnormalities, gastrointestinal symptoms, anemia, mineral and bone abnormalities, metabolic acidosis, cardiovascular complications and impaired immune function.

Diagnosis and Evaluation

The evaluation of suspected kidney disease begins with a detailed history and physical examination followed by laboratory assessment. Serum creatinine is commonly used to estimate glomerular filtration, while urine albumin-to-creatinine ratio provides important information regarding glomerular injury and prognosis. The combination of estimated glomerular filtration rate and albuminuria provides a more comprehensive assessment than either measure alone.[9]

The 2024 KDIGO CKD guideline emphasizes risk-based evaluation and management and recognizes the potential role of cystatin C in improving estimation of glomerular filtration in selected individuals.[9] Serial assessment is particularly important because a single abnormal result may not distinguish acute kidney injury from chronic kidney disease.

Urinalysis can identify hematuria, proteinuria, casts and other abnormalities that help direct further investigation. Imaging, particularly renal ultrasonography, is useful for assessing kidney size, structural abnormalities, cysts, hydronephrosis and obstruction. Serological investigations may be required when autoimmune, infectious or systemic causes are suspected. Kidney biopsy remains an important diagnostic procedure for selected glomerular and interstitial diseases when histopathological information will influence management.

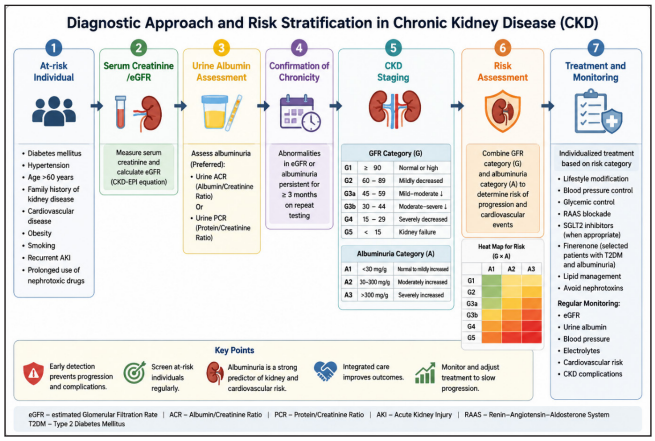

Figure 1.Diagnostic approach and risk stratification of patients with chronic kidney disease

Management of Kidney-Related Diseases

Management should be directed toward the underlying cause, prevention of further renal injury, reduction of cardiovascular risk and treatment of complications. In CKD, control of blood pressure, optimization of glycemic status, reduction of albuminuria, appropriate dietary management, smoking cessation and avoidance of nephrotoxins are important components of comprehensive care.[9]

Renin-angiotensin system inhibition remains an important therapeutic approach for patients with appropriate indications, particularly those with albuminuric CKD. The objective is not simply to normalize laboratory parameters but to reduce intraglomerular pressure, proteinuria and the risk of disease progression.

The emergence of SGLT2 inhibitors has significantly altered the therapeutic landscape of CKD. The DAPA-CKD trial demonstrated that dapagliflozin reduced the risk of kidney and cardiovascular outcomes in patients with CKD, including individuals without type 2 diabetes.[4] Similarly, the EMPA-KIDNEY trial demonstrated renal and cardiovascular benefits

with empagliflozin in a broad population of patients with CKD.[5] These findings have contributed to the incorporation of SGLT2 inhibitors into contemporary evidence-based CKD management for appropriate patients.

Nonsteroidal mineralocorticoid receptor antagonists have provided another therapeutic option, particularly for patients with type 2 diabetes and CKD. In the FIDELIO-DKD trial, finerenone reduced the risk of CKD progression and cardiovascular events compared with placebo, although monitoring for hyperkalemia remains important.[6] The FIGARO-DKD trial subsequently demonstrated cardiovascular benefits in patients with type 2 diabetes and CKD.[7]

Emerging evidence also supports consideration of combination approaches. Recent evidence from the CONFIDENCE trial evaluated simultaneous initiation of finerenone and empagliflozin in patients with CKD and type 2 diabetes, reflecting the movement toward multidrug cardiorenal protection rather than reliance on a single therapeutic pathway.[12] Such approaches require individualized assessment of renal function, potassium levels, blood pressure, volume status and potential drug interactions.

Management of Acute Kidney Injury

AKI requires rapid identification and correction of the precipitating cause. Management may involve restoration of circulating volume in appropriate patients, treatment of infection or sepsis, correction of hemodynamic abnormalities, relief of urinary obstruction and withdrawal or adjustment of nephrotoxic medications. Electrolyte and acid-base abnormalities should be monitored closely.

Kidney replacement therapy may become necessary in severe AKI when refractory hyperkalemia, severe metabolic acidosis, fluid overload, uremic complications or other clinically significant indications are present. Importantly, contemporary management increasingly emphasizes prevention and early recognition rather than treatment only after severe renal dysfunction has developed.[11]

Patients who recover from AKI require follow-up because recovery of serum creatinine does not necessarily indicate complete restoration of renal health. AKI is associated with an increased long-term risk of CKD, making post-AKI surveillance an important component of comprehensive kidney care.[11]

Kidney Failure and Renal Replacement Therapy

Progressive CKD may eventually lead to kidney failure, characterized by severely impaired renal function and accumulation of metabolic waste and fluid. At this stage, kidney replacement therapy or transplantation may be required depending on the patient's clinical status, comorbidities, preferences and suitability for transplantation.

Hemodialysis and peritoneal dialysis remain the principal dialysis modalities. Hemodialysis requires vascular access and extracorporeal blood purification, whereas peritoneal dialysis uses the patient's peritoneal membrane as a semipermeable membrane for solute and fluid exchange. Selection should consider medical suitability, lifestyle, home environment, access to services and patient preference.

Kidney transplantation generally provides the most comprehensive form of kidney replacement for eligible patients with kidney failure. However, transplantation is limited by donor availability, immunological considerations, surgical risks, long-term immunosuppression and healthcare-resource constraints.

Prevention and Public Health Approaches

Prevention represents a critical component of kidney disease control. Individuals at increased risk because of diabetes, hypertension, cardiovascular disease, obesity, family history or previous AKI should undergo appropriate renal assessment.[2] Early identification permits intervention before substantial irreversible renal damage develops.

Population-level strategies should emphasize control of diabetes and hypertension, reduction of tobacco exposure, promotion of healthy dietary patterns, physical activity and appropriate weight management. Rational use of medications and greater awareness of nephrotoxic exposures are also important.

Healthcare systems should incorporate kidney disease screening into existing chronic disease programs rather than treating nephrology as an isolated specialty. Integration of kidney assessment into diabetes and hypertension clinics can improve early detection. In resource-limited settings, low-cost blood and urine testing may provide an effective initial strategy for identifying individuals requiring further evaluation.

Emerging Developments in Nephrology

Contemporary nephrology is increasingly characterized by precision medicine, improved risk prediction and therapies targeting multiple mechanisms of disease progression. The 2024 KDIGO guideline incorporates individualized kidney failure risk assessment, greater attention to cystatin C and expanded use of SGLT2 inhibitors and other cardiorenal therapies.[9]

Digital health technologies may further transform kidney care. Electronic health-record algorithms, remote monitoring, home blood-pressure measurement and point-of-care testing can potentially improve detection and follow-up, particularly for patients living far from specialist centers. The emerging AKI/AKD framework also highlights

the potential role of biomarkers and electronic alerts in earlier recognition of kidney injury.[11]

Artificial intelligence and machine-learning methods may provide additional opportunities for prediction of CKD progression, AKI development and hospitalization. However, these approaches require careful validation, external testing, transparency and attention to healthcare inequities before widespread clinical implementation.

Genomic medicine is another developing field. Identification of genetic causes of unexplained CKD may improve diagnostic accuracy, facilitate family counseling and potentially guide disease-specific treatment. Similarly, advances in molecular biomarkers may help distinguish different renal disease phenotypes and identify patients most likely to respond to specific interventions.

Challenges in Kidney Disease Management

Despite major advances in diagnosis and treatment, substantial challenges remain. Underdiagnosis is one of the most important barriers because early CKD frequently produces no symptoms. Limited access to laboratory testing and specialist care can further delay diagnosis, particularly in rural and economically disadvantaged populations.

The financial burden associated with dialysis and transplantation represents another major challenge. Long-term dialysis requires substantial healthcare infrastructure, trained personnel, consumables and continuous patient support. Consequently, prevention and early-stage management are particularly important from both clinical and health-economic perspectives.

Another challenge is the growing complexity of patients with multimorbidity. Patients with CKD frequently have diabetes, hypertension, cardiovascular disease and multiple medications. Coordinated management is therefore essential to minimize polypharmacy, drug-related adverse events and inappropriate dosing.

Future Directions

Future kidney research should prioritize early detection, disease-specific biomarkers, precision therapeutics and strategies for reducing health inequities. The development of biomarkers capable of identifying kidney injury before substantial loss of glomerular filtration could transform clinical practice.

Therapeutic research is increasingly moving toward combinations that simultaneously target hemodynamic, metabolic, inflammatory and fibrotic pathways. The combination of renin-angiotensin system blockade, SGLT2 inhibition and selected mineralocorticoid receptor antagonism illustrates this evolving therapeutic philosophy.[6,9,12]

Research should also address kidney disease populations that remain underrepresented in clinical trials, including individuals from low- and middle-income countries, children, older adults with multimorbidity and patients with non-diabetic kidney diseases. Greater representation of diverse populations will improve the generalizability of future evidence.

Discussion

Kidney-related diseases represent a major and growing component of the global burden of non-communicable disease. Chronic kidney disease (CKD), acute kidney injury (AKI), glomerular disorders, hereditary kidney diseases and end-stage kidney disease collectively contribute to substantial morbidity, mortality and healthcare expenditure. The increasing recognition of CKD as a systemic disorder rather than an isolated renal abnormality has changed contemporary approaches to diagnosis and management. CKD is strongly associated with cardiovascular disease, diabetes, hypertension, anemia, mineral and bone abnormalities and premature mortality. The progressive nature of kidney dysfunction also means that early identification and intervention are essential to delay irreversible loss of renal function.

One of the most important observations emerging from current evidence is that kidney disease frequently remains clinically silent during its early stages. Patients may have substantial reductions in glomerular filtration or persistent albuminuria without experiencing prominent symptoms. Consequently, relying on clinical manifestations alone is inadequate for identifying individuals at risk. Estimation of glomerular filtration rate and assessment of urinary albumin excretion have therefore become fundamental components of CKD detection and staging. Persistent albuminuria is particularly important because it reflects glomerular and systemic vascular injury and is independently associated with progression of kidney disease and cardiovascular events. Early identification through appropriate laboratory assessment provides an opportunity for interventions before advanced renal dysfunction develops.

Diabetes mellitus and hypertension remain the principal drivers of CKD worldwide. Chronic hyperglycemia promotes glomerular hyperfiltration, endothelial dysfunction, oxidative stress and structural remodeling, eventually resulting in diabetic kidney disease. Similarly, prolonged elevation of blood pressure produces vascular and glomerular injury and accelerates nephron loss. The interaction between these conditions is particularly important because diabetes and hypertension frequently coexist, thereby producing cumulative renal injury. Effective management of glycemia and blood pressure is consequently central to preventing or slowing CKD progression.

The relationship between CKD and cardiovascular disease deserves particular emphasis. Declining renal function is associated with increased cardiovascular risk, and cardiovascular disease remains a major cause of death among individuals with CKD. Mechanisms underlying this association include hypertension, endothelial dysfunction, chronic inflammation, oxidative stress, anemia, vascular calcification and disturbances in mineral metabolism. Thus, CKD should be regarded as an important cardiovascular risk state. Management strategies should consequently extend beyond preservation of renal function and include comprehensive cardiovascular risk reduction.

AKI represents another important component of the kidney disease spectrum. Although AKI was historically considered a potentially reversible short-term event, evidence increasingly demonstrates that an episode of AKI may increase the subsequent risk of CKD, recurrent AKI and cardiovascular complications. The relationship between AKI and CKD is therefore bidirectional and clinically important. Patients who recover apparently normal serum creatinine levels after AKI may still require follow-up because structural or functional renal abnormalities may persist. Early recognition of AKI, correction of precipitating factors and appropriate post-AKI surveillance may therefore represent important opportunities for prevention of chronic renal impairment.

The development of newer pharmacological therapies has substantially changed the management of CKD. Sodium-glucose cotransporter-2 (SGLT2) inhibitors have emerged as important kidney-protective therapies because their benefits extend beyond glucose lowering. These agents can reduce intraglomerular pressure, slow decline in kidney function and reduce the risk of kidney failure and cardiovascular events in appropriately selected populations. Their expanding use demonstrates the transition from disease-specific treatment toward therapies that target shared mechanisms of renal and cardiovascular injury.

Mineralocorticoid receptor antagonism has also gained importance, particularly in patients with diabetic kidney disease. Finerenone, a non-steroidal mineralocorticoid receptor antagonist, has demonstrated renal and cardiovascular benefits in appropriately selected patients with diabetic CKD. Its therapeutic role highlights the importance of addressing inflammatory and fibrotic pathways in addition to conventional blood pressure and glycemic control. The increasing availability of complementary renoprotective therapies provides clinicians with opportunities to use multidrug strategies tailored to individual risk.

Renin-angiotensin system blockade remains an important component of treatment, particularly in patients with albuminuric CKD. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers can reduce intraglomerular pressure and albuminuria and slow progression of kidney disease in appropriate patients. Their use illustrates the importance of targeting hemodynamic mechanisms of glomerular injury. However, therapy must be accompanied by appropriate monitoring of serum creatinine and potassium, particularly in patients with advanced CKD or multiple medications affecting renal function.

Despite considerable advances, substantial challenges remain in the management of kidney disease. One major concern is the gap between evidence-based recommendations and implementation in routine clinical practice. Patients in low-resource settings may have limited access to screening, specialist care, laboratory testing and kidney replacement therapy. The burden of CKD is therefore not distributed equally, and socioeconomic factors can strongly influence diagnosis and treatment. Public-health strategies aimed at early detection, primary prevention and affordable access to essential medicines are essential to reduce these disparities.

Kidney replacement therapy represents a critical stage in the management of advanced kidney failure. Hemodialysis, peritoneal dialysis and kidney transplantation can prolong survival and improve quality of life, but access remains highly variable. Dialysis also imposes substantial financial and logistical burdens on patients and healthcare systems. Kidney transplantation generally offers important advantages for appropriately selected patients, but the availability of suitable organs, surgical infrastructure, immunosuppressive therapy and long-term follow-up limits accessibility in many regions.

Prevention therefore remains one of the most effective strategies for reducing the burden of kidney disease. Population-level interventions targeting diabetes, hypertension, obesity, smoking and inappropriate medication use may reduce the incidence of CKD. At the clinical level, periodic assessment of renal function and albuminuria in high-risk individuals can identify disease earlier. Patient education regarding medication adherence, blood pressure control, dietary habits and avoidance of nephrotoxic exposures can further contribute to renal protection.

Emerging technologies may provide additional opportunities for improving kidney care. Digital health platforms, electronic medical records, remote monitoring and artificial intelligence-based prediction models may facilitate earlier identification of individuals at high risk of renal deterioration. However, these technologies require rigorous validation and should complement rather than replace clinical assessment. Differences in healthcare infrastructure, digital literacy and access to technology must also be considered.

Overall, contemporary kidney care is moving from a predominantly reactive approach toward prevention, risk stratification and early intervention. The integration of laboratory biomarkers, cardiovascular risk assessment, novel pharmacological therapies, digital monitoring and multidisciplinary care has the potential to significantly alter the natural history of kidney disease. Nevertheless, the greatest benefits will depend on translating advances in nephrology into accessible, affordable and sustainable healthcare systems.

Conclusion

Kidney-related diseases encompass a broad spectrum of acute, chronic, structural, hereditary, glomerular, vascular and obstructive disorders and represent a major global health challenge. CKD is particularly important because it frequently progresses silently and is strongly associated with cardiovascular morbidity, kidney failure and premature mortality. Diabetes, hypertension, obesity, ageing, cardiovascular disease, previous AKI, autoimmune disease, genetic disorders, infections and nephrotoxic exposures contribute to the diverse epidemiology of renal disease.

Early identification through assessment of estimated glomerular filtration rate and albuminuria provides an opportunity to intervene before advanced kidney dysfunction develops. Contemporary management has progressed from conventional supportive treatment toward comprehensive cardiorenal protection involving renin-angiotensin system inhibition, SGLT2 inhibitors, mineralocorticoid receptor antagonists and individualized risk-based approaches. Evidence from major clinical trials has demonstrated clinically important benefits of dapagliflozin, empagliflozin and finerenone in appropriately selected populations.[4-7]

The future of nephrology is likely to involve increasingly personalized approaches integrating clinical risk assessment, biomarkers, genetics, digital health and novel therapeutics. Nevertheless, the greatest gains may continue to arise from relatively simple interventions: early detection, effective control of diabetes and hypertension, avoidance of kidney injury, appropriate medication use and equitable access to evidence-based care. A coordinated approach involving primary-care physicians, nephrologists, endocrinologists, cardiologists, nurses, dietitians and public-health professionals is essential to reduce the growing burden of kidney disease.

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