

Chronic Kidney Disease: Early Detection and Evidence-Based Management

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Abstract: Chronic kidney disease (CKD) has emerged as one of the fastest-growing causes of morbidity and mortality worldwide, affecting approximately one in ten adults and placing substantial pressure on healthcare systems. The condition is characterized by persistent abnormalities in kidney structure or function that progressively impair renal filtration, electrolyte homeostasis, endocrine activity, and cardiovascular health. Because CKD often remains clinically silent during its early stages, many patients are diagnosed only after irreversible nephron loss has occurred. Early recognition through systematic risk assessment, estimation of glomerular filtration rate, and detection of albuminuria provides an important opportunity to slow disease progression and reduce complications. Recent evidence has transformed CKD management by emphasizing individualized risk stratification, aggressive control of modifiable risk factors, optimization of blood pressure and glycemic management, and the incorporation of novel pharmacological therapies that provide both renal and cardiovascular protection. Advances in biomarker research, precision medicine, digital health technologies, and multidisciplinary care are further reshaping the management of chronic kidney disease. This review critically examines current concepts in CKD with particular emphasis on early detection, evidence-based therapeutic strategies, and future developments aimed at improving renal and cardiovascular outcomes.

Keywords: Chronic kidney disease; Albuminuria; Glomerular filtration rate; Kidney failure; Renal protection.

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Evidence-based management

Why Early Recognition Matters

Unlike many acute illnesses that present with obvious clinical manifestations, chronic kidney disease develops insidiously over many years. Functional reserve within the kidneys allows substantial nephron loss before symptoms become apparent, meaning that patients frequently remain unaware of progressive renal injury until advanced disease has developed. By the time fatigue, edema, reduced appetite, or uremic symptoms appear, a considerable proportion of renal function has often been permanently lost. Consequently, modern nephrology increasingly focuses on identifying CKD during its asymptomatic stages rather than waiting for overt clinical deterioration.

The global burden of CKD continues to rise because of increasing life expectancy and the growing prevalence of diabetes mellitus, hypertension, obesity, metabolic syndrome, and cardiovascular disease. In many regions, CKD has become a major contributor to disability-adjusted life years and healthcare expenditure owing to repeated hospitalization, cardiovascular complications, dialysis dependency, and kidney transplantation. Importantly, cardiovascular mortality among patients with CKD frequently exceeds the risk of progression to end-stage kidney disease, highlighting the close relationship between renal dysfunction and systemic vascular disease [1].

Current understanding recognizes CKD not simply as an isolated renal disorder but as a multisystem condition affecting cardiovascular, endocrine, hematological, skeletal, and metabolic function. Progressive nephron loss activates complex pathways involving inflammation, oxidative stress, endothelial dysfunction, fibrosis, and neurohormonal activation that contribute to worsening renal injury while simultaneously accelerating cardiovascular disease. This close interaction explains why patients with declining kidney function experience markedly increased risks of myocardial infarction, stroke, heart failure, peripheral arterial disease, and premature mortality compared with individuals with preserved renal function [2].

The major causes of CKD vary according to geographic region and socioeconomic development. Diabetes mellitus has become the leading cause of chronic kidney disease worldwide, followed closely by hypertension. Other important etiologies include chronic glomerular diseases, hereditary nephropathies, obstructive uropathy, autoimmune disorders, recurrent urinary tract infections, nephrotoxic medications, and congenital abnormalities of the kidneys and urinary tract. Regardless of the initiating cause, persistent renal injury ultimately converges into a common pathway characterized by glomerulosclerosis, tubular atrophy, interstitial fibrosis, and irreversible nephron loss.

The concept of kidney disease management has changed substantially during the past decade. Traditional care largely concentrated on delaying dialysis; contemporary practice instead emphasizes preserving kidney function, reducing cardiovascular complications, improving quality of life, and preventing hospitalization throughout every stage of disease. This broader perspective has encouraged closer collaboration between nephrologists, primary care physicians, cardiologists, endocrinologists, dietitians, and specialist nurses, recognizing that successful CKD management requires multidisciplinary care rather than isolated renal treatment.

Literature Review Approach

The evidence summarized in this review was obtained through a structured evaluation of contemporary scientific literature concerning chronic kidney disease. Searches were conducted using PubMed/MEDLINE, Scopus, Embase, Web of Science, and Google Scholar to identify peer-reviewed publications addressing epidemiology, diagnosis, prevention, and treatment of CKD. Particular emphasis was placed on studies published during the previous decade, although landmark investigations and international guideline documents were incorporated where they continue to influence current clinical practice.

Search terms included combinations of "chronic kidney disease," "glomerular filtration rate," "albuminuria," "renal fibrosis," "diabetic kidney disease," "hypertension," "SGLT2 inhibitors," "KDIGO guidelines," "renal replacement therapy," and "kidney protection." Randomized controlled trials, systematic reviews, meta-analyses, observational cohort studies, and evidence-based clinical guidelines were prioritized because of their methodological quality and clinical relevance. Publications were critically evaluated to synthesize current evidence regarding early diagnosis, disease progression, and therapeutic management.

Understanding the Evolution of Chronic Kidney Disease

The kidneys perform numerous physiological functions beyond filtration alone. They regulate body fluid volume, electrolyte balance, acid-base homeostasis, blood pressure, erythropoiesis, vitamin D metabolism, and removal of metabolic waste products. Progressive impairment of these diverse functions explains the broad systemic manifestations observed during CKD progression. Importantly, decline in renal function often occurs gradually, allowing adaptive physiological mechanisms to maintain biochemical stability until a significant proportion of nephron mass has already been lost.

Nephron injury initiates a cascade of compensatory responses within the remaining functional glomeruli. Surviving nephrons undergo hypertrophy and hyperfiltration in an attempt to preserve overall renal function. Although initially beneficial, persistent hyperfiltration increases intraglomerular pressure, promotes proteinuria, and accelerates glomerular sclerosis. Progressive activation of inflammatory pathways, oxidative stress, transforming growth factor-beta signaling, and fibroblast proliferation ultimately leads to irreversible renal fibrosis. Once extensive fibrosis develops, restoration of normal kidney architecture becomes unlikely despite treatment, emphasizing the importance of early intervention before structural damage becomes permanent [3].

Several clinical conditions accelerate this pathological process. Chronic hyperglycemia induces glomerular basement membrane thickening, mesangial expansion, and diabetic nephropathy through metabolic and inflammatory mechanisms. Long-standing hypertension causes vascular remodeling, nephrosclerosis, and ischemic nephron injury, while obesity contributes through insulin resistance, chronic inflammation, and increased renal workload. Recurrent exposure to nephrotoxic medications, autoimmune diseases, inherited renal disorders, and persistent urinary tract obstruction further increase susceptibility to progressive kidney damage.

From a clinical perspective, CKD should not be viewed solely according to declining glomerular filtration rate. Current international classification integrates estimated glomerular filtration rate (eGFR) with urinary albumin excretion because albuminuria reflects ongoing glomerular injury and provides important prognostic information regarding both renal and cardiovascular outcomes. Patients with preserved filtration but significant albuminuria may possess greater long-term risk than individuals with moderately reduced filtration alone, highlighting the importance of comprehensive renal assessment rather than reliance upon serum creatinine measurements alone.



Risk factors for CKD can broadly be categorized into non-modifiable and modifiable determinants. Increasing age, genetic predisposition, family history of kidney disease, congenital abnormalities, and certain ethnic backgrounds represent fixed risk factors. Conversely, diabetes mellitus, hypertension, obesity, smoking, dyslipidemia, sedentary lifestyle, recurrent acute kidney injury, excessive dietary sodium intake, and prolonged exposure to nephrotoxic agents constitute potentially reversible contributors. Recognition and modification of these factors provide the greatest opportunity for preventing disease progression before irreversible nephron loss occurs.

Rather than representing an inevitable consequence of aging, chronic kidney disease should therefore be regarded as a largely preventable chronic disorder when risk factors are identified early and managed aggressively. Contemporary preventive nephrology increasingly emphasizes systematic screening of high-risk populations, timely diagnosis, and implementation of kidney-protective interventions before advanced structural damage develops.

Early Detection and Diagnostic Evaluation

Early detection remains the most effective strategy for reducing the long-term consequences of chronic kidney disease because therapeutic interventions are substantially more successful before irreversible nephron loss and extensive renal fibrosis develop. Since most individuals remain asymptomatic during the initial stages of CKD, routine screening should focus on populations with established risk factors rather than the general population. Patients with diabetes mellitus, hypertension, cardiovascular disease, obesity, autoimmune disorders, recurrent urinary tract infections, nephrolithiasis, family history of kidney disease, or previous episodes of acute kidney injury represent groups in whom periodic renal assessment provides considerable clinical benefit [4].

The diagnosis of CKD is established when abnormalities in kidney structure or function persist for at least three months and have implications for overall health. Contemporary diagnostic criteria extend beyond reduced glomerular filtration alone and include persistent albuminuria, abnormalities detected by imaging studies, histopathological changes, electrolyte disturbances caused by tubular dysfunction, or structural abnormalities involving the kidneys and urinary tract. This broader definition reflects the recognition that clinically important kidney disease may be present even when filtration remains relatively preserved [5].

Serum creatinine continues to be the most commonly used laboratory marker for estimating kidney function; however, interpretation of isolated creatinine concentrations may be misleading because values are influenced by age, sex, muscle mass, ethnicity, nutritional status, and certain medications. Consequently, estimated glomerular filtration rate (eGFR) has become the preferred method for evaluating renal function because it provides a more accurate assessment of filtration capacity after adjustment for demographic variables. Serial measurement of eGFR is considerably more informative than a single value because progressive decline over time more accurately reflects disease progression than isolated laboratory results.

Assessment of urinary albumin excretion has become equally important in contemporary nephrology. Persistent albuminuria reflects glomerular injury, predicts progression toward kidney failure, and independently increases cardiovascular risk. Measurement of the urine albumin-to-creatinine ratio (ACR) using an early morning urine specimen has therefore become the preferred screening method because of its convenience, reproducibility, and prognostic significance. The combination of eGFR and albuminuria provides substantially greater risk stratification than either parameter alone and now forms the basis of international CKD classification systems [5].

Table 1. Initial Evaluation of Patients with Suspected Chronic Kidney Disease

Assessment Component	Recommended Investigation	Clinical Purpose
Kidney Function	Serum creatinine and estimated GFR	Evaluate filtration capacity
Proteinuria	Urine albumin-to-creatinine ratio (ACR)	Detect glomerular injury
Urinalysis	Dipstick and microscopic examination	Identify hematuria, proteinuria, infection
Blood Pressure	Standardized measurement	Identify hypertension and disease progression
Blood Tests	Electrolytes, glucose, HbA1c, lipid profile	Detect metabolic abnormalities and risk factors
Renal Imaging	Kidney ultrasonography	Evaluate kidney size, obstruction, and structural abnormalities

Clinical evaluation should extend beyond laboratory investigations and include a detailed medical history focused on duration of hypertension or diabetes, exposure to nephrotoxic medications, herbal preparations, previous episodes of acute kidney injury, recurrent urinary tract infections, autoimmune disease, and family history of inherited nephropathies. Physical examination should evaluate blood pressure, body mass index, evidence of fluid overload, peripheral edema, vascular disease, and signs of systemic disorders that may contribute to renal dysfunction.



Although laboratory testing provides essential information regarding renal function, imaging studies frequently identify structural abnormalities responsible for chronic kidney disease. Renal ultrasonography remains the preferred initial imaging modality because it is non-invasive, widely available, and capable of detecting reduced kidney size, cortical thinning, hydronephrosis, congenital abnormalities, renal cysts, obstructive uropathy, and asymmetrical renal disease. Doppler ultrasonography may additionally evaluate renal blood flow when renovascular disease is suspected.

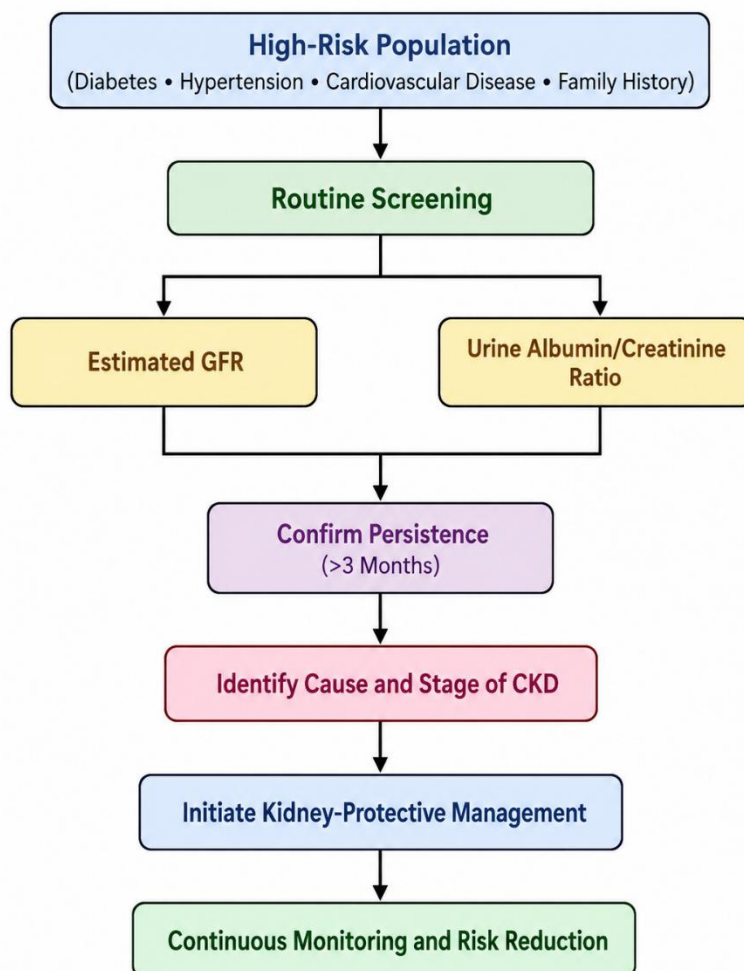


Figure 1. Framework for Early Detection of Chronic Kidney Disease

A practical framework illustrating the sequential approach to early detection of chronic kidney disease in high-risk individuals. Confirmation of persistent abnormalities allows timely intervention before advanced renal impairment develops.

Additional laboratory investigations should be guided by the suspected underlying cause of kidney disease. Autoimmune serology, complement levels, viral screening, serum and urine protein electrophoresis, genetic testing, and renal biopsy may be indicated in selected patients presenting with rapidly progressive renal impairment, unexplained proteinuria, persistent hematuria, or suspected glomerular disease. Renal biopsy remains the definitive diagnostic procedure for many primary glomerular disorders because histopathological findings directly influence prognosis and therapeutic selection.

Increasing attention has also focused on the role of novel biomarkers capable of identifying kidney injury before conventional laboratory abnormalities become apparent. Molecules such as kidney injury molecule-1 (KIM-1), neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, and urinary tubular injury markers have demonstrated promising diagnostic potential in research settings. Although these biomarkers have not yet replaced traditional renal function testing in routine clinical practice, they may facilitate earlier diagnosis and more accurate prediction of disease progression in the future [6].

Accurate staging of CKD requires integration of kidney function, albuminuria, underlying etiology, and associated comorbidities. Rather than serving merely as a classification system, staging provides important prognostic information regarding progression to kidney failure, cardiovascular risk, frequency of follow-up, and timing of specialist referral. This multidimensional assessment enables clinicians to individualize monitoring strategies and initiate appropriate interventions according to each patient's overall risk profile rather than relying solely on declining glomerular filtration.

Evidence-Based Management

Management of chronic kidney disease extends far beyond preservation of renal filtration alone. Contemporary treatment aims to delay disease progression, prevent cardiovascular complications, correct metabolic abnormalities, improve quality of life, and postpone or avoid the need for renal replacement therapy. Because CKD frequently coexists with diabetes mellitus, hypertension, cardiovascular disease, and metabolic disorders, successful management requires coordinated multidisciplinary care rather than isolated nephrological intervention. Individualized treatment plans should consider the underlying etiology, stage of kidney disease, degree of albuminuria, cardiovascular risk, and associated comorbid conditions [7].

Blood pressure control remains the single most effective intervention for slowing CKD progression in both diabetic and non-diabetic populations. Persistent hypertension accelerates glomerular injury by increasing intraglomerular pressure and promoting progressive nephrosclerosis. Current clinical guidelines recommend individualized blood pressure targets while emphasizing standardized measurement techniques and long-term treatment adherence. Agents that inhibit the renin–angiotensin–aldosterone system, including angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), remain the preferred first-line therapies in patients with albuminuria because they reduce intraglomerular pressure, decrease urinary protein excretion, and provide significant renal protection beyond blood pressure reduction alone [8].

Optimal glycemic control represents another fundamental component of CKD management, particularly among individuals with diabetic kidney disease. Sustained hyperglycemia accelerates glomerular injury through oxidative stress, advanced glycation end-product formation, inflammation, and microvascular damage. Individualized glycemic targets should balance prevention of chronic complications against the increased risk of hypoglycemia observed in advanced kidney disease. Recent evidence has demonstrated that sodium-glucose cotransporter-2 (SGLT2) inhibitors significantly reduce progression of chronic kidney disease while simultaneously lowering cardiovascular morbidity and mortality. These medications have therefore become an integral component of contemporary kidney-protective therapy irrespective of diabetic status in many patients [9].

Nutritional management also contributes substantially to slowing disease progression and minimizing metabolic complications. Dietary sodium restriction improves blood pressure control and reduces fluid retention, while excessive protein intake should generally be avoided because it increases glomerular hyperfiltration and metabolic burden. Individualized nutritional counseling should ensure adequate caloric intake while preventing protein-energy wasting, electrolyte imbalance, and micronutrient deficiencies. Patients with advanced CKD frequently require adjustment of dietary potassium and phosphorus intake according to biochemical findings and stage of disease [10].

Management of dyslipidemia remains essential because cardiovascular disease represents the leading cause of death among individuals with CKD. Statin therapy effectively reduces major cardiovascular events and should be considered in most adults with non-dialysis-dependent CKD who possess elevated cardiovascular risk. Smoking cessation, regular physical activity, maintenance of healthy body weight, moderation of alcohol intake, and avoidance of nephrotoxic medications further contribute to comprehensive cardiovascular and renal protection [11].

Table 2. Evidence-Based Interventions for Slowing CKD Progression

Therapeutic Strategy	Primary Target	Expected Clinical Benefit
Blood pressure optimization	Hypertension	Slows nephron loss and reduces cardiovascular events
ACE inhibitors or ARBs	Albuminuria and RAAS activation	Reduces proteinuria and preserves kidney function
SGLT2 inhibitors	Diabetic and non-diabetic CKD	Delays CKD progression and lowers cardiovascular risk
Glycemic control	Diabetes mellitus	Prevents diabetic nephropathy progression
Dietary sodium reduction	Fluid overload and hypertension	Improves blood pressure and reduces edema
Lifestyle modification	Multiple cardiovascular risk factors	Improves long-term renal and cardiovascular outcomes



Correction of CKD-associated complications is equally important during long-term follow-up. Progressive decline in renal function frequently results in anemia, mineral and bone disorders, metabolic acidosis, hyperkalemia, and fluid overload. Renal anemia is primarily caused by reduced erythropoietin production and iron deficiency and may require iron supplementation or erythropoiesis-stimulating agents according to current treatment recommendations. Disturbances in calcium, phosphorus, vitamin D metabolism, and parathyroid hormone regulation should also be identified early because untreated mineral disorders contribute to vascular calcification, skeletal complications, and increased cardiovascular mortality [12].

Patients approaching advanced stages of CKD require careful preparation for renal replacement therapy. Timely referral to nephrology services enables appropriate education regarding dialysis modalities, vascular access creation, transplantation options, and conservative supportive care where appropriate. Early specialist involvement has consistently been associated with improved survival, reduced emergency dialysis initiation, and better preparation for long-term treatment decisions.

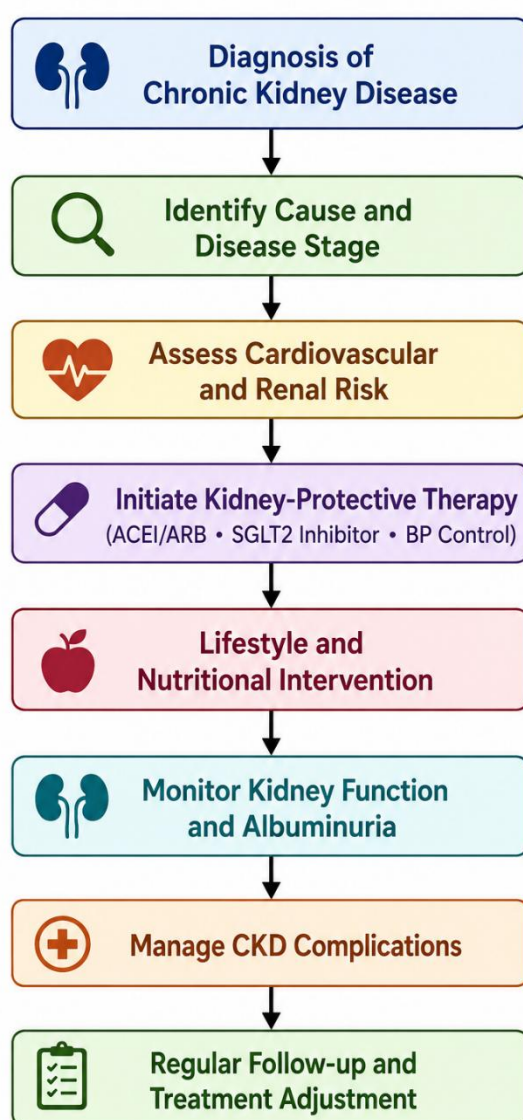


Figure 2. Comprehensive Management Pathway for Chronic Kidney Disease

Integrated management strategy demonstrating progression from diagnosis and risk assessment to pharmacological treatment, lifestyle modification, complication management, and preparation for renal replacement therapy when indicated.

Advances in digital medicine are increasingly influencing CKD care. Electronic clinical decision-support systems, telemedicine, wearable monitoring devices, and remote blood pressure surveillance allow closer follow-up while reducing unnecessary hospital visits. Artificial intelligence models capable of predicting rapid renal function decline and identifying patients at highest risk for kidney failure are currently under investigation and may facilitate earlier intervention in routine clinical practice. These technologies have particular relevance for patients residing in remote regions where access to specialist nephrology services remains limited.

Table 3. Emerging Developments in Chronic Kidney Disease Care

Innovation	Current Status	Potential Clinical Impact
Artificial intelligence risk prediction	Clinical evaluation	Earlier identification of rapid CKD progression
Novel renal biomarkers	Research and selective clinical use	Earlier detection of kidney injury
Precision medicine	Expanding investigation	Individualized treatment strategies
Tele-nephrology	Increasing implementation	Improved access and long-term monitoring
Wearable health technologies	Early adoption	Continuous assessment of blood pressure and physiological parameters
Regenerative therapies	Experimental	Potential restoration of renal tissue in future

Rather than focusing exclusively on declining kidney function, contemporary CKD management emphasizes preservation of overall health through integrated cardiovascular protection, prevention of complications, optimization of metabolic control, and individualized long-term follow-up. This comprehensive approach has substantially improved patient outcomes and continues to evolve with advances in pharmacotherapy, precision medicine, and digital healthcare.

DISCUSSION

Chronic kidney disease has become a major global health concern because of its progressive nature, close relationship with cardiovascular disease, and increasing prevalence among aging populations. Unlike many acute renal disorders, CKD develops gradually over many years, often without producing recognizable symptoms until substantial nephron loss has occurred. Consequently, delayed diagnosis remains one of the principal barriers to successful treatment. The evidence reviewed in this article demonstrates that systematic screening of high-risk individuals, combined with early implementation of kidney-protective therapies, can substantially delay disease progression and reduce both renal and cardiovascular complications [1].

A notable shift in modern nephrology has been the transition from treating advanced renal failure to preventing progressive nephron loss at much earlier stages of disease. Current clinical practice recognizes that kidney damage frequently begins long before significant reductions in glomerular filtration become apparent. Albuminuria, hypertension, endothelial dysfunction, and low-grade inflammation often precede measurable decline in renal function and therefore provide valuable opportunities for early therapeutic intervention. This preventive approach has fundamentally altered management strategies, emphasizing continuous risk assessment rather than waiting for severe renal impairment to develop [5].

Blood pressure optimization remains one of the most effective interventions for slowing CKD progression regardless of the underlying cause. Long-term studies consistently demonstrate that inhibition of the renin-angiotensin-aldosterone system reduces intraglomerular hypertension, decreases proteinuria, and preserves residual kidney function. More recently, the emergence of sodium-glucose cotransporter-2 inhibitors has represented one of the most important therapeutic advances in nephrology. Originally introduced for diabetes management, these agents have shown significant renal and cardiovascular benefits even among patients without diabetes, leading to substantial changes in international treatment recommendations [9].

Equally important is the recognition that chronic kidney disease should not be managed as an isolated renal disorder. Cardiovascular disease remains the leading cause of death among individuals with CKD, and progressive renal impairment frequently coexists with hypertension, diabetes mellitus, obesity, dyslipidemia, anemia, and mineral bone disorders. Effective management therefore requires comprehensive cardiovascular risk reduction together with correction of metabolic abnormalities and preservation of kidney function. This integrated approach has become the cornerstone of contemporary CKD care and highlights the importance of multidisciplinary collaboration involving nephrologists, primary care physicians, cardiologists, endocrinologists, dietitians, pharmacists, and specialist nurses [7].

Lifestyle interventions continue to complement pharmacological therapy throughout every stage of CKD. Dietary sodium restriction, smoking cessation, weight management, regular physical activity, avoidance of nephrotoxic medications, and



individualized nutritional counseling contribute significantly to slowing disease progression while improving cardiovascular health. However, maintaining long-term adherence remains challenging for many patients because dietary modifications, multiple medications, and frequent clinical follow-up often create considerable treatment burden. Structured patient education and shared decision-making therefore remain essential components of successful long-term management [10].

Technological innovation is expected to further improve early diagnosis and long-term monitoring. Electronic health records integrated with automated risk prediction models, artificial intelligence algorithms, tele-nephrology services, and wearable monitoring devices may facilitate earlier identification of patients at greatest risk for rapid renal decline. Novel biomarkers capable of detecting tubular injury before conventional biochemical abnormalities become apparent may further improve diagnostic precision and enable more timely intervention. Although many of these technologies remain under evaluation, they represent promising developments for future clinical practice [13].

Despite substantial progress, several challenges remain unresolved. Access to nephrology services, affordability of novel medications, delayed referral, limited public awareness, and disparities in healthcare delivery continue to contribute to poor outcomes, particularly in low-resource settings. Furthermore, current therapies slow rather than reverse established renal fibrosis, emphasizing the need for continued investigation into regenerative medicine, anti-fibrotic therapies, and personalized treatment strategies capable of restoring kidney structure and function.

Future Perspectives

Future management of chronic kidney disease is expected to become increasingly individualized through integration of molecular diagnostics, precision medicine, and digital healthcare technologies. Advances in genomic research, transcriptomics, proteomics, and metabolomics are improving understanding of disease heterogeneity and may allow earlier identification of patients predisposed to rapid disease progression or poor therapeutic response.

Artificial intelligence has emerged as an important area of investigation within nephrology. Predictive algorithms capable of analyzing laboratory trends, imaging findings, clinical characteristics, and electronic health records may improve risk stratification and support individualized treatment planning. Integration of these technologies into routine clinical practice may facilitate earlier intervention while reducing avoidable hospitalization and progression to kidney failure.

Research into regenerative medicine also continues to expand. Stem cell therapies, tissue engineering, anti-fibrotic agents, and novel molecular therapies targeting inflammatory and fibrotic pathways may eventually provide opportunities to restore renal tissue rather than merely delaying disease progression. Although these approaches remain experimental, they represent promising directions for future nephrology research.

Expansion of tele-nephrology and remote monitoring systems is also expected to improve long-term follow-up, particularly for patients residing in geographically underserved regions. Improved accessibility to specialist care, combined with patient-centered digital education and home monitoring technologies, may significantly enhance treatment adherence and clinical outcomes.

CONCLUSION

Chronic kidney disease remains a major cause of morbidity, mortality, and healthcare utilization worldwide. Because the disease frequently progresses silently during its early stages, timely recognition through systematic screening of high-risk individuals remains essential for preserving renal function and reducing cardiovascular complications. Contemporary diagnostic strategies integrating estimated glomerular filtration rate, albuminuria assessment, laboratory evaluation, and renal imaging have significantly improved early detection and risk stratification.

Current evidence supports a comprehensive management strategy centered on blood pressure optimization, renin-angiotensin system inhibition, sodium-glucose cotransporter-2 inhibitors, individualized glycemic control, lifestyle modification, and proactive management of CKD-associated complications. Multidisciplinary care, patient education, nutritional intervention, and regular monitoring further contribute to slowing disease progression and improving quality of life.

Future advances in precision medicine, artificial intelligence, novel biomarkers, and regenerative therapies are expected to refine diagnosis and expand treatment opportunities. Nevertheless, improving global outcomes will require equitable access to evidence-based therapies, greater public awareness, earlier referral to nephrology services, and continued investment in preventive healthcare. Through early detection and integrated long-term management, the growing burden of chronic kidney disease can be substantially reduced.



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