

CHRONIC KIDNEY DISEASE: PATHOGENESIS, CLINICAL FEATURES AND MODERN MANAGEMENT

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Abstract

Chronic Kidney Disease (CKD) is a progressive and irreversible decline in renal function lasting longer than three months. It represents a major global health burden due to its association with cardiovascular morbidity, end-stage renal disease (ESRD), and increased mortality. This study analyzes the pathogenesis, risk factors, diagnostic criteria, clinical manifestations, complications, and current treatment approaches of CKD.

Keywords: Chronic Kidney Disease, CKD, Glomerular Filtration Rate, Nephropathy, Hypertension, Diabetes Mellitus, Renal Failure, Dialysis, Proteinuria, Nephron Loss

Introduction

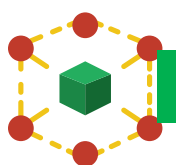
Chronic Kidney Disease affects approximately 10–13% of the global population. It is defined as abnormalities of kidney structure or function present for more than three months, with implications for health.

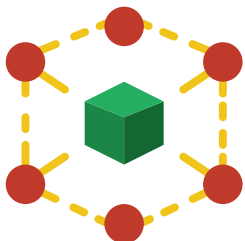
CKD commonly develops as a complication of:

- Type 2 Diabetes Mellitus
- Hypertension
- Glomerulonephritis
- Polycystic kidney disease
- Autoimmune disorders

The kidneys play essential roles in:

- Filtration of metabolic waste
- Regulation of electrolytes





- Acid-base balance
- Blood pressure regulation
- Erythropoietin production

Progressive nephron loss leads to reduced Glomerular Filtration Rate (GFR) and accumulation of toxic metabolites in the bloodstream.

The aim of this study is to analyze the pathophysiology, progression stages, clinical manifestations, and modern management strategies of CKD.

Methods

This study is based on systematic analysis of peer-reviewed journals, KDIGO guidelines (Kidney Disease: Improving Global Outcomes), WHO reports, and major nephrology clinical trials published between 2015–2024.

Inclusion criteria:

- Adults ≥ 18 years
- Diagnosed CKD (Stage 1–5)
- Studies with ≥ 300 participants

Exclusion criteria:

- Acute kidney injury
- Pediatric nephropathy
- Transient renal impairment

Data were evaluated focusing on:

- GFR decline rate
- Albuminuria levels
- Cardiovascular outcomes
- Dialysis requirement

Results

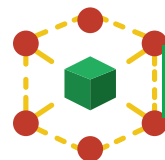
1. Epidemiological Findings

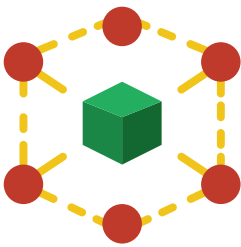
- CKD prevalence: ~10% worldwide
- Diabetes accounts for 40% of CKD cases
- Hypertension contributes to 25–30%
- Higher incidence in elderly population

2. Pathophysiology

CKD progression involves:

Step 1: Initial Nephron Injury





Damage to glomeruli due to hyperglycemia or hypertension.

Step 2: Compensatory Hyperfiltration

Remaining nephrons increase workload.

Step 3: Progressive Fibrosis

Chronic inflammation leads to interstitial fibrosis and nephron loss.

Figure 1. Nephron Damage in Chronic Kidney Disease (Description)

The illustration shows glomerular sclerosis, thickened basement membrane, and narrowing of capillary lumen. Fibrotic tissue replaces normal nephron structure, reducing filtration capacity.

3. CKD Staging (Based on GFR)

- Stage 1: GFR ≥ 90 (Kidney damage with normal function)
- Stage 2: GFR 60–89
- Stage 3: GFR 30–59
- Stage 4: GFR 15–29
- Stage 5: GFR < 15 (End-Stage Renal Disease)

4. Clinical Manifestations

Early CKD is often asymptomatic.

Advanced symptoms include:

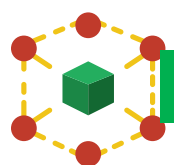
- Fatigue
- Edema
- Hypertension
- Decreased urine output
- Uremic symptoms (nausea, confusion)

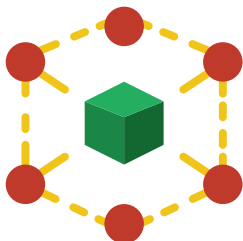
Figure 2. Systemic Effects of Chronic Kidney Disease (Description)

The diagram demonstrates how reduced kidney function leads to fluid retention, electrolyte imbalance (hyperkalemia), anemia (due to low erythropoietin), and cardiovascular strain.

5. Laboratory Findings

- Elevated serum creatinine
- Reduced GFR
- Proteinuria (albuminuria)
- Electrolyte imbalance
- Metabolic acidosis





Persistent albuminuria is an early marker of kidney damage.

6. Complications

Cardiovascular Disease

CKD significantly increases risk of myocardial infarction and stroke.

Anemia

Reduced erythropoietin production leads to normocytic anemia.

Bone-Mineral Disorder

Impaired vitamin D metabolism causes hypocalcemia and secondary hyperparathyroidism.

Figure 3. CKD-Mineral Bone Disorder Mechanism (Description)

The illustration shows decreased vitamin D activation, phosphate retention, and increased parathyroid hormone levels leading to bone demineralization.

7. Treatment Strategies

Lifestyle Modification

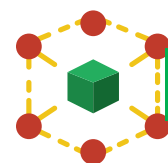
- Sodium restriction (<2g/day)
- Blood pressure control
- Glycemic control in diabetics
- Smoking cessation

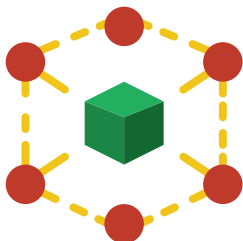
Pharmacological Therapy

1. ACE inhibitors / ARBs
 - Reduce proteinuria
 - Slow CKD progression
2. SGLT2 inhibitors
 - Provide nephroprotective effects
3. Erythropoiesis-stimulating agents
 - Treat anemia
4. Phosphate binders
 - Manage mineral imbalance

Renal Replacement Therapy

- Hemodialysis
- Peritoneal dialysis
- Kidney transplantation





Discussion

CKD is a progressive condition driven by chronic inflammation, oxidative stress, and glomerular hypertension. Early detection through screening of high-risk populations (diabetics, hypertensive patients) is crucial.

Modern management focuses on:

- Slowing GFR decline
- Reducing cardiovascular risk
- Preventing complications
- Improving quality of life

SGLT2 inhibitors have emerged as a breakthrough therapy due to their significant renal protective effects beyond glucose control.

Public health strategies should prioritize early screening and preventive nephrology care.

Conclusion

Chronic Kidney Disease is a silent but progressive internal disease with serious systemic consequences. Early diagnosis, strict blood pressure control, glycemic management, and modern nephroprotective therapy significantly delay progression to end-stage renal disease. Comprehensive patient-centered care remains essential.

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