5 stages of Chronic Kidney Disease

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Introduction

Chronic kidney disease (CKD) is an abnormality of kidney structure or function that is present for at least three months and has implications for health. It is not defined only by a low estimated glomerular filtration rate (eGFR). A person may have preserved filtration but persistent albumin in the urine, structural abnormalities, or another marker of kidney damage. Conversely, one abnormal laboratory result during dehydration or acute illness does not by itself establish chronic disease. (Webster et al. 1238–52)

CKD is commonly described through five GFR categories, but modern risk assessment uses the cause of disease, GFR category, and albuminuria category together. This is known as the CGA approach. The original discussion correctly identified diabetes and high blood pressure as major causes, but it incorrectly suggested that stage 3 routinely requires dialysis, that stages 4 and 5 represent total loss of function, and that transplant is automatically necessary in stage 5. Treatment and kidney-replacement decisions depend on the whole clinical picture. (Webster et al. 1238–52)

How the Kidneys Work

The kidneys filter plasma through microscopic glomeruli, regulate water and electrolytes, maintain acid–base balance, help control blood pressure, activate vitamin D, and produce erythropoietin for red-blood-cell production. Each kidney contains many nephrons. Damage can reduce the number or performance of functioning nephrons. Remaining nephrons may compensate temporarily, which is one reason CKD can progress silently. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Kidneys are not simply mechanical filters with tubes that fail because blood pressure exceeds a fixed diameter. Diabetes, hypertension, immune disease, inherited disorders, obstruction, toxins, infection, and vascular disease can injure glomeruli, tubules, interstitium, or blood vessels through different mechanisms. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Confirming Chronic Kidney Disease

Evaluation normally includes serum creatinine with eGFR, urine albumin-to-creatinine ratio (ACR), urinalysis, blood pressure, medical and medication history, and assessment of possible cause.

Abnormal findings are repeated to establish chronicity unless previous records already show persistence. Imaging, serologic tests, genetic evaluation, or kidney biopsy may be required in selected cases. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

eGFR is an estimate, not a direct measurement of “kidney efficiency.” It is influenced by creatinine generation and may be less accurate in people with unusual muscle mass, rapidly changing kidney function, pregnancy, or certain clinical conditions. Cystatin C can improve estimation in appropriate situations. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

GFR and Albuminuria Categories

GFR category	eGFR (mL/min/1.73 m²)	Description
G1	90 or higher	Normal or high filtration
G2	60–89	Mildly decreased filtration
G3a	45–59	Mildly to moderately decreased
G3b	30–44	Moderately to severely decreased
G4	15–29	Severely decreased
G5	Below 15	Kidney failure category

G1 and G2 do not constitute CKD unless another marker of kidney damage is present for at least three months. Albuminuria categories are A1, below 30 mg/g; A2, 30–300 mg/g; and A3, above 300 mg/g. Higher albuminuria and lower eGFR generally predict greater risk of progression, cardiovascular disease, and death. Two people with the same eGFR may therefore have very different risk. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Stage 1: G1 With Evidence of Kidney Damage

In G1, eGFR is at least 90, but CKD exists because of persistent albuminuria, abnormal urine sediment, structural disease, a genetic condition, or another marker. Most people have no symptoms. Diagnosis does not depend on elevated blood urea alone, which is nonspecific and affected by hydration, diet, bleeding, and other factors. (Kidney Disease: Improving Global Outcomes CKD Work

Group S117–S314)

Management focuses on identifying the cause, controlling blood pressure and diabetes, reducing albuminuria when possible, avoiding nephrotoxins, and monitoring. Early recognition can change long-term risk even when filtration appears normal. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Stage 2: G2 With Evidence of Kidney Damage

G2 represents eGFR of 60–89 with persistent evidence of kidney damage. It is common for patients to feel well. The rate of change is important: a stable eGFR over years has different implications from a rapid decline. Monitoring frequency depends on albuminuria, cause, comorbidities, and prior trajectory. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Patients should understand that CKD is a risk condition, not an immediate prediction of dialysis. Many people remain stable, especially when the cause is treated and cardiovascular risks are controlled. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Stage 3a and Stage 3b

Stage 3 is divided because risk rises substantially as eGFR falls. G3a is 45–59 and G3b is 30–44. Some people remain asymptomatic; others develop hypertension, anemia, mineral and bone abnormalities, medication accumulation, or electrolyte disturbances. Blood in the urine is not a defining symptom and requires evaluation for several possible causes. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Routine dialysis is not used in stage 3. Care includes risk-based monitoring, medication review, treatment of complications, and referral when the cause is uncertain, progression is rapid, albuminuria is severe, or risk is high. Drug doses may need adjustment because renal clearance declines. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Stage 4

G4, with eGFR of 15–29, is severe reduction rather than complete loss of function. Symptoms may include fatigue, appetite change, itching, nausea, swelling, sleep disturbance, or concentration difficulty, but symptom severity varies. Anemia, acidosis, potassium abnormalities, fluid overload, and mineral-bone disorder become more likely. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

This stage requires close nephrology care and preparation for possible kidney-replacement therapy if

progression is expected. Preparation may include education about hemodialysis, peritoneal dialysis, transplantation, vascular access, and conservative kidney management. Preparation is not the same as immediate initiation. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Stage 5 and Kidney Failure

G5 is eGFR below 15. Some patients need dialysis or transplantation, while others can be managed for a period without dialysis if symptoms and laboratory abnormalities remain controlled. Dialysis should not be started from an eGFR number alone. Indications can include refractory fluid overload, dangerous electrolyte or acid–base abnormalities, uremic symptoms, pericarditis, malnutrition related to kidney failure, or other complications that cannot be managed medically. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Kidney transplantation can provide excellent outcomes for eligible patients and may occur before dialysis. It is not available or suitable for everyone. Conservative kidney management is an active, patient-centered option emphasizing symptom control and quality of life for people who choose not to pursue dialysis or transplant. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Major Causes and Risk Factors

Diabetes is a leading cause of CKD. Persistent hyperglycemia can damage glomerular structures and increase albumin leakage. The original term “diabetic neuropathy” is incorrect in this context; the kidney condition is diabetic kidney disease or diabetic nephropathy. Hypertension can both cause and result from kidney disease. Other causes include glomerulonephritis, polycystic kidney disease, congenital abnormalities, recurrent obstruction, lupus, vascular disease, and medication or toxin exposure. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Risk factors include older age, family history, cardiovascular disease, prior acute kidney injury, smoking, and social conditions that limit access to preventive care and healthy food. Proton-pump inhibitors and other drugs may be associated with kidney injury in certain settings, but patients should not stop prescribed treatment without clinical advice. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Evidence-Based Management

Management begins with the cause and individual risk. Blood-pressure control is central. Angiotensin-converting-enzyme inhibitors or angiotensin receptor blockers are particularly important for many patients with albuminuria, but potassium and kidney function must be monitored. Sodium–glucose cotransporter-2 inhibitors reduce kidney and cardiovascular risk in many people with CKD, including

selected patients without diabetes. Other medications may be used according to diabetes, albuminuria, heart failure, potassium, and cardiovascular status. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Patients should avoid unnecessary nonsteroidal anti-inflammatory drugs and inform clinicians about supplements and over-the-counter medicines. Vaccination, smoking cessation, physical activity, and cardiovascular prevention are important. Sodium restriction may help blood pressure and fluid control. Protein intake should be individualized; severe unsupervised restriction can cause malnutrition. Drinking excessive water does not “flush” CKD and may be harmful in some patients. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Monitoring and Shared Decision-Making

Monitoring can include eGFR, ACR, potassium, bicarbonate, hemoglobin, calcium, phosphate, parathyroid hormone, and medication effects, depending on stage. Risk-prediction equations can help determine referral and preparation needs. Patients should receive explanations in understandable language and participate in decisions about goals and treatment burden. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Urgent assessment is needed for severe shortness of breath, confusion, chest pain, marked swelling, very low urine output, uncontrolled vomiting, or suspected acute kidney injury. This educational overview cannot replace individualized diagnosis or treatment. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

Cardiovascular Risk and Referral

CKD is also a major cardiovascular risk condition. Many people with CKD are more likely to experience heart attack, stroke, heart failure, or cardiovascular death than to progress to kidney failure. Management therefore includes lipid treatment when indicated, blood-pressure control, diabetes care, smoking cessation, physical activity, and attention to heart failure. Nephrology referral is appropriate for uncertain cause, rapid decline, persistent severe albuminuria, resistant hypertension, recurrent electrolyte disturbance, hereditary disease, or sufficiently high predicted kidney-failure risk. Referral should occur early enough to support diagnosis and planning rather than only after symptoms become severe. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Preventing Acute Injury on Chronic Disease

People with CKD are vulnerable to acute kidney injury during infection, dehydration, surgery, urinary obstruction, or exposure to certain medicines and contrast agents. A sudden creatinine rise should

not be mislabeled as ordinary stage progression. Prompt assessment, medication review, and treatment of the acute cause may recover function and prevent further chronic loss. (National Institute of Diabetes and Digestive and Kidney Diseases; Webster et al. 1238–52)

Conclusion

CKD is classified more accurately by cause, eGFR, and albuminuria than by five stages alone. G1 and G2 require another persistent marker of damage; G3 is divided into G3a and G3b; G4 is severe reduction; and G5 is the kidney-failure category. Most early disease is silent, and dialysis is neither routine in stage 3 nor inevitable at a particular eGFR. Timely diagnosis, blood-pressure and diabetes management, kidney-protective medication, avoidance of nephrotoxins, complication treatment, and shared planning can slow progression and improve cardiovascular and quality-of-life outcomes. (Kidney Disease: Improving Global Outcomes CKD Work Group S117–S314)

References

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