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Chronic Kidney Disease



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Section 1: Introduction

The kidneys are small organs, about the size of a standard computer mouse, yet they play a major role in keeping the body functioning. In addition to their well-known role in filtering blood to remove waste, toxins, and excess fluid, the kidneys have other important functions. They play a key role in regulating blood pressure. They signal the body to produce red blood cells and are important for maintaining bone health. The kidneys also regulate essential chemicals in the blood. These small, yet mighty, organs filter all of the blood in the body every thirty minutes and are necessary to sustain life (CDC, 2024).

When the kidneys are unable to function properly, the effects extend throughout the entire body. Chronic kidney disease (CKD), a condition in which kidney function gradually declines over time, is a major public health concern and one of the leading causes of death in the United States. An estimated 35.5 million U.S. adults have CKD, yet approximately 40% of individuals with severely reduced kidney function are unaware they have the disease (CDC, 2024). The burden of CKD continues to grow, with the condition now ranking as the ninth leading cause of death worldwide, with deaths increasing by 95% between 2000 and 2021 (Shirsat et al., 2025).

Nurses are an important member of the healthcare team when caring for those with CKD. Nurses who are aware of the signs and symptoms of early CKD can make a significant impact on outcomes through early recognition. When CKD is recognized early, interventions can slow the progression of kidney function decline, significantly affecting longevity and quality of life. To recognize early CKD, nurses should be knowledgeable about renal anatomy and normal nephron function. They must also understand the various stages of CKD, risk factors, and cardiovascular implications. Nurses should be able to interpret lab results related to CKD and be knowledgeable about the medications and procedures used to

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treat the condition. Effective nursing interventions and patient education can help slow the progression of CKD and improve patient outcomes.

Section 2: How do the kidneys work?

In most human bodies, there are two kidneys, which are bean-shaped, retroperitoneal, located on each side of the spine, and positioned at the level between the T12 and L3 vertebrae (Garza & Leslie, 2025), just below the rib cage in the lower back. Each kidney drains into a ureter that leads to the bladder (Cleveland Clinic, 2025). Typically, the left kidney is slightly larger than the right, and male kidneys are usually larger than female kidneys. The size of kidneys can vary from person to person, but on average, they are typically each 10-12 cm long, 5-7 cm wide, and 3-5 cm thick. Each kidney is enclosed by a two-layered capsule, referred to as the renal capsule, and then further protected by layers of fat, often referred to as the Gerota fascia (Garza & Leslie, 2025).

Approximately 20-25% of cardiac output is directed to the kidneys, which is about 1,200 mL/min of renal blood flow. This equates to 600 mL/min of renal plasma flow. As blood flows into the kidney, approximately 20% passes into the renal tubules. This number is known as the glomerular filtration rate, which is approximately 120 mL/min or 180 L/day in an average adult (Madrazo-Ibarra & Vaitla, 2023).

Each kidney contains two primary regions: the renal parenchyma and the pyelocalyceal (collecting) system. The renal parenchyma contains the cortex and the medulla, and the pyelocalyceal system contains minor and major calyces and the renal pelvis. The cortex is located in the outer portion of the renal parenchyma and contains most of the components of the nephrons (Garza & Leslie, 2025). This is where blood filtration begins and where the hormone erythropoietin is produced (Cleveland Clinic,2025). The renal mantle is the layer of

the cortical tissue that is at the base of each renal pyramid, and the renal columns are the parts of the cortical tissue that extend from the mantle and towards the renal sinus on each side of the renal pyramids (Garza & Leslie, 2025). The renal sinus is in the middle part of the kidney. It is lined with fat to protect the structures it contains, including nerves, arteries, veins, lymphatic channels, and the pyelocalyceal system (Cleveland Clinic, 2022). Between the cortex and renal sinus is the medulla. The medulla is organized into renal pyramids. Each renal pyramid consists of rays of tissue, known as medullary rays, which extend from the cortex. At the apex of each renal pyramid is a renal papilla, the structure from which urine drains. Each renal pyramid and its surrounding cortex is considered a renal lobe, and each kidney contains an average of nine renal lobes (Garza & Leslie, 2025).

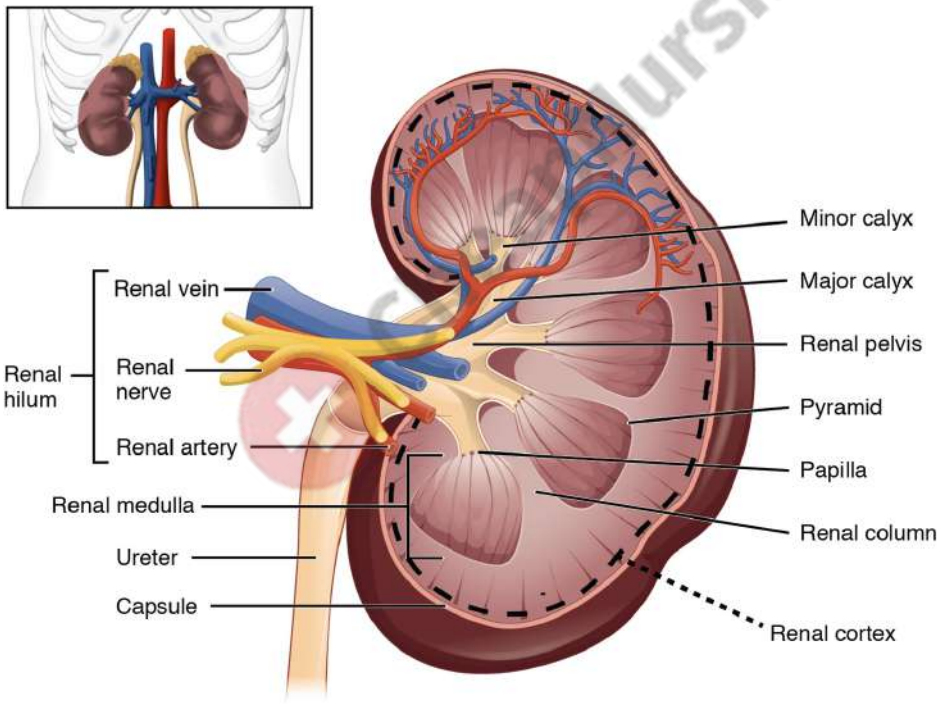


Figure 1. Macroscopic Anatomy of the Left Kidney. (Bach et al., 2026)

The renal collecting system collects the urine produced in renal parenchyma through a series of ducts and drains it through the urinary system to the bladder for excretion. The renal papilla of each pyramid will drain into a minor calyx. In some cases, the renal papillae of smaller pyramids may drain into the same minor calyx, forming a compound calyx. A few of the minor calyces, usually 2-3, then merge to form a major calyx. Multiple major calyces merge to form the renal pelvis. The renal pelvis is funnel-shaped and transitions into the proximal ureter, where urine drains into the bladder (Garza & Leslie, 2025).

The nephron is where the kidney's primary function, filtration, occurs. Each adult kidney contains 1-1.5 million nephrons, but there can be more. This microscopic structure contains a glomerulus and a complex tubular system. The proximal convoluted tubule (PCT), which is the first portion of this complex tubular system, and the glomerulus are in the renal cortex. The loop of Henle, shaped like a hairpin, is a portion of this structure that extends through the medulla and then returns to the cortex, where it transitions into the distal convoluted tubule. From here, the nephron drains into the collecting duct. There are two types of nephrons: superficial and juxtamedullary (Madrazo-Ibarra & Vaitla, 2023). Superficial nephrons make up 85% of all nephrons (Kingston et al., 2024) and have glomeruli near the cortical surface. The loop of Henle of these nephrons is short. The juxtamedullary nephrons make up the other 15% of nephrons and have glomeruli that are located close to the cortico-medullary junction. These nephrons have long loops of Henle that penetrate deeper into the medulla (Madrazo-Ibarra & Vaitla, 2023). The juxtamedullary nephrons have larger glomeruli and can achieve higher glomerular filtration rates (Kingston et al., 2024).

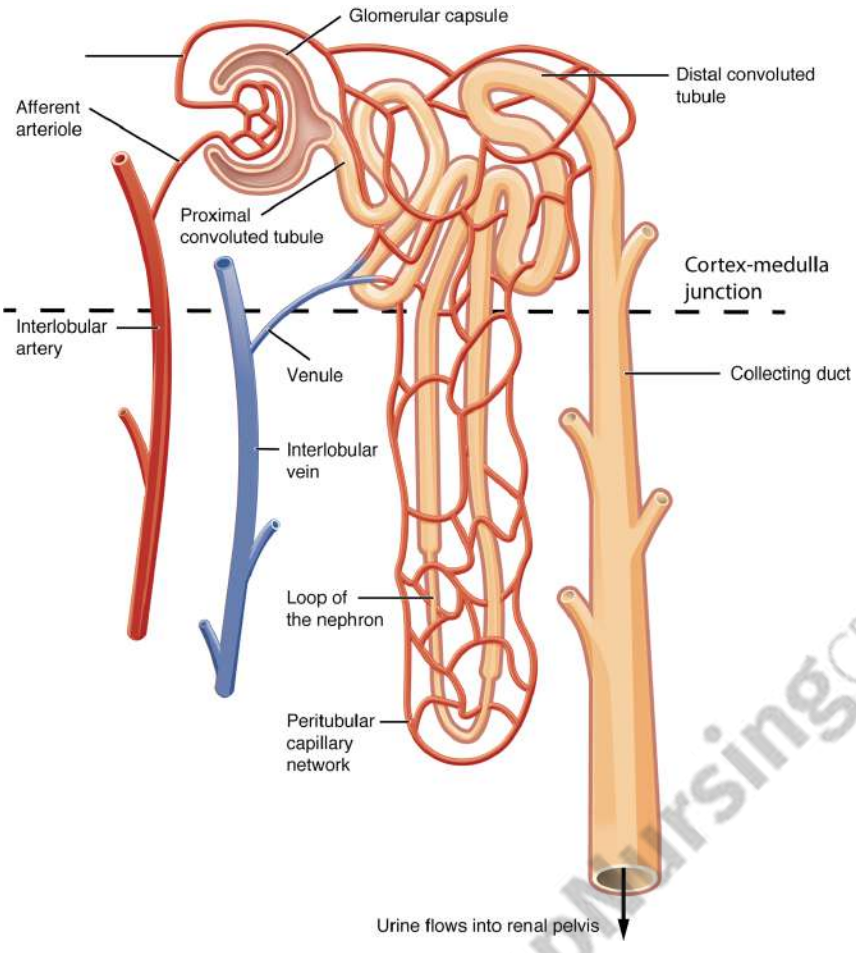


Figure 2. Blood flow in the nephron. (Bach et al., 2026)

It helps to understand how blood is filtered by understanding how it flows through the kidney and into each nephron. Blood enters the kidneys through the renal arteries, which then branch until each glomerulus is supported by a single afferent arteriole. This vessel delivers blood to the glomerular capillaries. The efferent arteriole transports blood from the glomerulus to the peritubular capillaries before the blood returns to venous circulation through a venule that leads to an interlobular vein (Kingston et al., 2024).

Urine production begins in the glomerulus through a process called glomerular filtration. (Kingston et al., 2024). The glomerulus is a small tuft of capillaries that is protected by a covering called the glomerular capsule or Bowman's capsule, a double-layered epithelial structure (Garza & Leslie, 2025). Together, the

glomerulus and Bowman's capsule are often referred to as the renal corpuscle (Kingston et al., 2024). The afferent and efferent arterioles are located next to the tuft of capillaries and are responsible for regulating the pressure within the glomerulus. The capillaries of the glomerulus are unique in that they can filter large volumes of blood using a specialized filtration barrier. After the blood is filtered, the ultrafiltrate, or fluid and substances that have been filtered from the blood, moves into the space between the filtration barrier and Bowman's capsule before flowing into the proximal convoluted tubule (PCT) (Madrazo-Ibarra & Vaitla, 2023).

The PCT is responsible for absorbing and transporting water, electrolytes, and other solutes as they come into contact with the tubule epithelium. While the kidneys produce 160-180L of ultrafiltrate per day, only 1.5-2 L of urine are excreted daily. The PCT reabsorbs 60-65% of water and most of the electrolytes and nutrients contained in the ultrafiltrate. A complex network of capillaries that surrounds the PCT, known as the peritubular capillaries, reabsorbs free water and other plasma components. As the PCT transitions to the Loop of Henle, the structure dilutes the urine through active sodium transport, reabsorbing 30-40% of sodium (Madrazo-Ibarra & Vaitla, 2023).

The loop of Henle is comprised of three parts, which are the thin descending limb, thin ascending limb, and a thick ascending limb. The thin descending limb narrows as it crosses into the renal medulla. As it ascends back towards the renal cortex, the tube widens, hence the term "thick ascending limb". The thin descending limb is highly permeable to water, and water is reabsorbed through osmosis at this point. The thin ascending limb and thick ascending limb are not permeable to water, but permeable to solutes. This is where urine is diluted. Water that is reabsorbed is returned to circulation through the renal vasa recta (Madrazo-Ibarra & Vaitla, 2023).

The juxtaglomerular apparatus is a cluster of specialized cells located alongside the afferent arteriole of the glomerulus. This structure regulates glomerular filtration. It is a cluster of cells that includes the macula densa cells of the thick ascending limb within the cortex and smooth muscle cells of the afferent arteriole. The thick ascending limb transitions to the DCT as it returns to the renal cortex near the glomerulus and then transitions to the cortical collecting tubule. The DCT reabsorbs filtered sodium chloride and secretes potassium via various cellular mechanisms (Madrazo-Ibarra & Vaitla, 2023).

The connecting tubules are the final part of the nephron. They are composed of two different types of cells: intercalated cells and connecting tubule cells. The intercalated cells regulate the secretion of hydrogen and bicarbonate. The connecting tubule leads to the collecting tubule, where it converges with connecting tubules of other nephrons and drains into a collecting duct. The structure then transitions into the collecting tubules, which are the end of the nephron and where urine drains into a collecting duct (Madrazo-Ibarra & Vaitla, 2023).

Section 2 Personal Reflection

What is the primary function of the kidneys? What is glomerular filtration rate? What does this number indicate? Where does blood filtration begin? What is the nephron? Where does urine go once it leaves the nephron?

Section 3: What is Chronic Kidney Disease?

Chronic kidney disease is defined as “abnormalities of kidney structure or function, present for a minimum of three months, with implications for health” (Stevens et al., 2024). Specifically, this is determined by an estimated glomerular

filtration rate (eGFR) of less than 60mL/min/1.73m2 throughout the three-month period (Braga et al., 2022). The term “chronic kidney disease” may also be used to describe when there is at least one significant marker of kidney damage, which may include albuminuria, urine sediment abnormalities, persistent hematuria, electrolyte abnormalities due to tubular disorders, histological and structural abnormalities, and/or history of kidney transplantation (Stevens et al., 2024).

Chronic kidney disease is progressive, marked by an increasing loss in renal function, often eventually leading to the need for renal replacement therapy (RRT) (Vaidya & Aeddula, 2024). This progressive loss of function is due to a decline in the number of functioning nephrons, limiting the kidneys' ability to filter metabolic waste and balance electrolytes (Braga et al., 2022). When the kidneys are not functioning adequately, there are extensive systemic impacts on cardiovascular health, cognitive function, and bone metabolism that can lead to many conditions, including hypertension, anemia (Vaidya & Aeddula, 2024), increased risk for infections, alterations in electrolytes, changes in appetite, and lower quality of life (CDC, 2024).

Since CKD affects multiple organ systems, the signs and symptoms of CKD are varied. In the very early stages of CKD, individuals may not have any symptoms, or they may attribute symptoms to other conditions. Early symptoms may include foamy urine, polyuria, itchy or dry skin, fatigue, nausea, decreased appetite, and unintentional weight loss. While symptoms may be subtle at first, they tend to worsen significantly as the disease progresses. Symptoms of more severe CKD include poor concentration, numbness or swelling of the extremities, muscle aches, shortness of breath, vomiting, sleep disturbances, and breath that smells like ammonia, often described as “fishy” or smelling like urine. In cases of advanced CKD, patients may also experience symptoms of cardiovascular disease, worsened hypertension, anemia, metabolic acidosis, mineral and bone disorders, hyperkalemia, and kidney failure (National Kidney Foundation,2023b).

Chronic kidney disease begins on a microscopic level. The kidneys are well-equipped to repair themselves after acute injuries, which can be caused by dehydration or hypotension; however, they are not as well-equipped to repair multiple cumulative injuries. When multiple microinjuries occur over time, the healing response of the kidney, producing interstitial fibroblasts to restore structure, progresses to the point where functional tissue is replaced by connective tissue. This condition, known as fibrosis, can occur in different areas of the nephron. The primary causes of this progressive decline in function are diabetes mellitus, hypertension, metabolic disorders, sepsis, ischemia, reperfusion injuries, and exposure to nephrotoxins (Braga et al., 2022).

Since CKD is progressive, it is helpful to use a classification system to describe the current state of kidney function. Multiple classification systems are used simultaneously to describe the patient's clinical condition accurately. CKD is categorized based on cause, glomerular filtration rate, and level of albuminuria (Stevens et al., 2024). This is often referred to as CGA (cause, GFR, albuminuria) staging (National Kidney Foundation, 2026). The causes of CKD vary widely, so it is important to include this when describing a case of CKD, as it provides information on the likely structural changes that are contributing to the decline in kidney function and helps the healthcare team individualize management (Stevens et al., 2024). Standardized classifications also improve communication among members of the healthcare team.

There are six GFR-related categories (G1-G5), with G3 split into two distinct levels: G3a and G3b. Each GFR category is defined by a specific GFR range that indicates the degree of kidney function impairment. It is important to note that if the GFR falls into G1 or G2, the patient is not considered to have CKD unless there is additional evidence of kidney damage (National Kidney Foundation, 2026). For patients with CKD, categories G1 and G2 describe kidneys that have some damage but continue to function well. Stage G3a is the most common CKD stage found in

patients and is typically when symptoms of CKD begin to present. Stage 3b describes kidneys that are not functioning well, but treatment can often delay or prevent CKD from progressing beyond this stage. Stage G4 describes when there is some kidney function, but it is poor due to severe damage. Category G5 occurs when the kidneys are close to failure or have already failed. At this stage, renal replacement therapy is indicated (Cleveland Clinic, 2023).

GFR Categories of Chronic Kidney Disease

Category	GFR (mL/min/1.73 m^2)	Terms
G1	≥ 90	Normal or High
G2	60-89	Mildly decreased*
G3a	45-59	Mildly to moderately decreased
G3b	30-44	Moderately to severely decreased
G4	15-29	Severely decreased
G5	<15	Kidney failure

*Relative to young adult level (National Kidney Foundation,2026)

Albuminuria is considered one of the earliest signs of kidney dysfunction (Barzilay et al., 2024). There are three levels of albuminuria to describe the clinical status of a patient with CKD (National Kidney Foundation,2026). Albuminuria, also called proteinuria, occurs when albumin is present in the urine. While albumin is an important protein in the blood essential for muscle building, tissue repair, and immune responses, it is typically not found in urine. However, small quantities can be considered normal. When the kidneys are functioning properly, albumin cannot pass through the glomerular filters (National Kidney Foundation, 2023a). Therefore, albuminuria is a sign of structural damage in the glomerulus (Barzilay

et al., 2024). An individual can have albuminuria with a normal GFR (National Kidney Foundation, 2023a). Evaluating the presence and amount of albumin in the urine is one method for testing for albuminuria, typically using an early-morning urine sample. (Vaidya & Aeddula, 2024). Albuminuria is measured using a urinary albumin-creatinine ratio (ACR). Typically, creatinine is filtered into the urine, while albumin is not, so this provides very specific information about the health of the glomerular filters. When the glomerulus is damaged, both albumin and creatinine will pass into the urine. Two or more elevated results within three months indicate a likely diagnosis of kidney disease (National Kidney Foundation,2023b).

Albuminuria Categories of Chronic Kidney Disease

Category	ACR (mg/g)	Terms
A1	<30	Normal to mildly increased
A2	30-300	Moderately increased*
A3	>300	Severely increased

*Relative to young adult level (National Kidney Foundation,2026)

Categories of albuminuria are based on the ACR in milligrams of albumin per gram of creatinine. An ACR of less than 30 is considered normal to mildly increased and is considered category A1. Category A2 is defined as an ACR from 30-300, indicating moderately increased albuminuria. Category A3 describes albuminuria greater than 300 and indicates severely increased albuminuria (National Kidney Foundation, 2026). Since the introduction of this classification system in 2012 by Kidney Disease: Improving Global Outcomes (KDIGO), there have been improvements in the identification of CKD. It should be noted, however, that this

system can potentially lead to overdiagnosis of CKD, especially in older adults (Vaidya & Aeddula, 2024).

Chronic kidney disease can also be described by identifying the physiological or anatomical point at which the kidneys are no longer functioning well. Prerenal disease occurs when renal perfusion is decreased. Prolonged impairment of kidney perfusion can cause acute tubular necrosis, leading to progressive loss of kidney function. One potential cause of prerenal disease may be prolonged hypotension. Intrinsic renal disease describes when pathology of the vessels, glomeruli, or tubules is contributing to poor kidney function. Postrenal disease occurs due to an obstruction of the ureters that does not allow urine to drain from the kidneys. An obstruction could be due to prostatic disease, nephrolithiasis (kidney stone), a tumor exerting pressure on the ureter, or a congenital condition, such as neurogenic bladder (Vaidya & Aeddula, 2024).

Section 3 Personal Reflection

What is CKD? Why do you think CKD is progressive? How is a classification system for CKD used among members of the healthcare team? Why are multiple classification systems used? What do GFR and albumin-to-creatinine ratio reveal about renal function?

Section 4: Risk Factors and Cardiovascular Implications

There are many potential causes of CKD, and the risk factors for developing the disease are often the same as those for conditions that lead to CKD. Though some risk factors for CKD may be genetic, others are modifiable. Screening for risk factors related to CKD is part of routine primary care. Nurses must be

knowledgeable about risk factors for CKD development and for disease progression in individuals already diagnosed with the condition (WHO, 2026).

Many conditions can lead to chronic kidney disease. The most common cause of CKD is Type 2 diabetes, contributing to 30-50% of all cases of CKD. This is followed by hypertension, which accounts for 27.2% of all cases. Primary glomerulonephritis accounts for approximately 8.2% of CKD cases. Other conditions that frequently lead to CKD include Type 1 diabetes, chronic tubulointerstitial nephritis, hereditary or cystic diseases, secondary glomerulonephritis or vasculitis, plasma cell dyscrasias or neoplasm, and sickle cell nephropathy (Vaidya & Aeddula, 2024).

Some risk factors for CKD cannot be modified. This includes advanced age, male sex, and non-White ethnicity. Black Americans, Afro-Caribbean individuals, Hispanics, and Asians are all at increased risk for CKD and CKD progression. There are also specific heritable genes that are associated with diabetic nephropathy and CKD progression. Multiple gene variants in the general population can increase the risk of renal scarring and affect the renin-angiotensin-aldosterone system (RAAS). This hormonal pathway regulates blood pressure and fluid balance, thereby influencing CKD progression (Vaidya & Aeddula, 2024). Individuals over age 60 are at increased risk for CKD. A family history of CKD also increases risk. Those who have a history of acute kidney injury are at increased risk due to prior damage to the nephrons. Inherited conditions, such as polycystic kidney disease, can also contribute to a decline in kidney function due to anatomical variations (National Kidney Foundation, 2023b).

Many risk factors can be modified to reduce the risk of developing CKD. Risk factors for developing CKD include diabetes, hypertension, cardiovascular disease, and obesity (National Kidney Foundation, 2023b). It is estimated that 1 in 3 individuals with diabetes also have CKD (CDC, 2024). Chronic hyperglycemia

damages the microvasculature of the nephron, leading to kidney dysfunction. CKD and diabetes mellitus have a bidirectional relationship, meaning they each exacerbate the other. While elevated blood sugar contributes to diabetic nephropathy, a decline in kidney function limits the kidney's ability to regulate glucose levels by selectively reabsorbing glucose as needed. This decline in glucose metabolism can lead to or worsen insulin resistance and diabetes (Kumar et al., 2023). Hyperfiltration associated with diabetes mellitus due to insulin resistance can lead to glomerulosclerosis. Elevated insulin levels also exert an anti-lipolytic effect, contributing to further metabolic dysregulation (Dong & Tong, 2025).

Hypertension is the second leading cause of CKD, and approximately 1 in 5 adults with hypertension has CKD (CDC, 2024). Hypertension causes narrowing of blood vessels, reducing blood flow and weakening them. This includes the blood vessels in the kidneys, which become less able to filter waste and excess fluid from the cardiovascular system. Like diabetes, hypertension also has a bidirectional relationship with CKD. As hypertension causes a decline in the kidney's ability to filter out extra fluid, the extra fluid present in the body further increases blood pressure. Individuals with heart failure have an increased risk of CKD, and those with CKD are also at increased risk for heart failure. Cardiovascular disease also limits perfusion to the kidneys (CDC, 2024).

Obesity, defined as having a body mass index (BMI) of 30 or higher, is a significant risk factor for developing CKD. This is because obesity significantly increases the risk of diabetes and hypertension, which are the primary risk factors for developing CKD (CDC, 2024). Obesity is an independent risk factor for albuminuria, even before a rise in creatinine. Obesity is also known to contribute to the progression of CKD. Increased adipose tissue, especially visceral fat, activates multiple metabolic pathways that lead to chronic inflammation, damaging the kidneys' blood vessels and impairing glomerular filtration. Obesity

also contributes to insulin resistance. Hyperinsulinemia is known to lead to increased glomerular hyperfiltration and increased vascular permeability. It also leads to salt and water retention, leading to hypertension. Ectopic renal fat can also compress the blood vessels of the kidneys, which causes perfusion problems and increased activation of the RAAS, leading to hypertension. While obesity is often associated with cardiovascular disease, it contributes to CKD in many other ways independent of cardiovascular disease (Shirsat et al., 2025).

The primary risk factors for disease progression in those who have already been diagnosed with CKD are systemic hypertension, albuminuria, and metabolic factors. Systemic hypertension increases blood pressure in the glomerular capillary beds. This condition is known as glomerular hypertension, which can lead to glomerulosclerosis. Increased systolic blood pressure, specifically, is a reliable predictor of CKD progression. Elevations in systolic blood pressure are associated with complications for those with CKD. In both individuals with diabetic kidney disease and those with nondiabetic kidney disease, the presence of albuminuria is associated with a faster rate of CKD progression. Metabolic factors that increase the risk for CKD development and progression include insulin resistance, dyslipidemia, and hyperuricemia (Vaidya & Aeddula, 2024).

Other comorbid health conditions can lead to CKD or contribute to disease progression. Diseases that affect the glomerulus, such as glomerulonephritis, IgA nephropathy (IgAN), and HIV nephropathy, compromise renal function. Lupus, an autoimmune condition, can lead to lupus nephritis, in which the immune system attacks the kidneys, damaging them and causing renal dysfunction. Severe infections, such as those leading to sepsis and hemolytic uremic syndrome, can damage the kidneys. Other conditions that can contribute to kidney dysfunction include kidney cancer, kidney stones, frequent or untreated urinary tract infections, hydronephrosis, and congenital abnormalities of the kidney and urogenital system (National Kidney Foundation, 2023b).

Acute kidney injury, previously known as acute kidney failure, occurs when the kidneys abruptly lose the ability to filter blood. This can occur very quickly, in days or even just hours. There are many potential causes of acute kidney injury. These can include other health conditions that increase stress on the kidneys, such as pregnancy, cancer, severe infections, hypotension, anaphylaxis, and severe burns. Certain medications, including higher doses of non-steroidal anti-inflammatory drugs (NSAIDs), as well as recreational drugs and toxic substances, can cause acute injury to the kidneys. When an individual has experienced an acute kidney injury, they are at increased risk for developing CKD (National Kidney Foundation, 2024).

Smoking is a risk factor for many significant health conditions, and CKD is no exception. Although research results on the relationship between smoking and glomerular filtration rate and albuminuria vary, there is an established link between smoking and oxidative stress, inflammation, and RAAS activation, leading to vascular damage and renal fibrosis. Smoking also has indirect effects on kidney function, as it increases the risk of cardiovascular disease, hypertension, type 2 diabetes, and obesity, which all have direct links to CKD. Research suggests that those who smoke have a 20% increased risk of developing CKD when compared to nonsmokers. Individuals who have CKD and smoke are at four times greater risk for progression to end-stage renal disease (Yang et al., 2025).

How does CKD affect the cardiovascular system?

There are cardiac implications related to CKD. Cardiovascular disease is a risk factor for CKD, but it can also be a consequence of CKD (De Bhailis & Kalra, 2022). Individuals with CKD are at increased risk for developing cardiovascular disease, including coronary artery disease, stroke, heart failure, arrhythmias, and sudden cardiac death. A diagnosis of CKD has a multifactorial effect on the cardiovascular

system. In addition to the typical risk factors associated with both conditions, including hypertension, diabetes, and dyslipidemia, there are CKD-specific conditions that contribute to cardiovascular disease. Inflammation, oxidative stress, activation of the RAAS, fluid overload, alterations in hemodynamics, mineral and bone disorders, accumulation of uremic toxins, and CKD-related changes that occur in protein structures can all lead to adverse structural changes in the heart and blood vessels. Calcification of the vasculature is a common finding in CKD due to hemodynamic changes and dysregulated mineral metabolism. Proinflammatory mediators increase as CKD progresses, causing inflammation that affects the cells of the vessel walls. The cardiac changes that occur with CKD lead to fibrosis and cardiac hypertrophy, markers of uremic cardiomyopathy. It is estimated that 1/3 of patients with CKD have left ventricular hypertrophy. This number grows as CKD progresses. The interactions between CKD and cardiovascular disease are complex and significantly affect patients with both conditions (Marx-Schütt et al., 2025).

Hypertension resulting from CKD can have implications for the entire cardiovascular system. Research has found evidence to suggest that individuals in stages 3 or 4 of CKD are at significantly increased risk of cardiovascular death. When there are a reduced number of functioning nephrons, there is an increased response from the sympathetic nervous system and RAAS. Upregulation of the RAAS increases intravascular volume. Increased sympathetic nervous system activity leads to hypertension, which can eventually lead to left ventricular hypertrophy if it goes untreated. As CKD advances, endothelial dysfunction becomes more prevalent. This causes stenosis in the arteries, contributing to hypertension and increased risk of adverse cardiovascular events. As CKD progresses, tissues become increasingly damaged by hypoxia, leading to oxidative stress, an inflammatory response, and eventually renal fibrosis (De Bhailis & Kalra,

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2022). This risk of cardiovascular complications for those with CKD is further elevated if the individual also has diabetes (Kumar et al., 2023)

When someone has been diagnosed with CKD, it is important that they understand there are strategies to reduce the risk of disease progression by focusing on modifiable risk factors. Managing hypertension and albuminuria through medications, dietary changes, and weight loss can slow the progression of CKD. For those with albuminuria and diabetic kidney disease, the use of RAAS blockers can slow progression. Smoking cessation and weight loss are effective strategies to reduce risk of progression. Achieving a BMI within an optimal range can also improve metabolic factors that lead to CKD progression, including insulin resistance, dyslipidemia, and hyperuricemia (Vaidya & Aeddula, 2024).

Section 4 Personal Reflection

What are non-modifiable risk factors for CKD? What are modifiable risk factors? How do diabetes and hypertension affect the kidneys? Why is obesity considered a risk factor? What cardiovascular implications are present when a patient is diagnosed with CKD?

Section 5: Laboratory Interpretation

In addition to understanding how GFR and albumin-to-creatinine ratios are used to stage CKD, they should also understand how these tests can be affected by factors such as the calculation method used. Nurses must also understand how other laboratory tests are used to assess renal function and guide treatment. By understanding best practices for sample collection and what factors may alter test results, nurses can ensure accurate and useful test results. When nurses understand what different lab values indicate to the healthcare team about the

patient's condition, they can determine whether treatment is effective and anticipate any changes needed in the care plan. Early symptoms of chronic kidney disease can be subtle, but changes in bloodwork values may occur before symptoms become obvious. When nurses are aware of what changes in lab values mean, they can help to improve outcomes.

Nurses must understand the timing and collection methods that best yield accurate results, since lab results often guide the treatment plan and nursing interventions. When assessing renal function, the optimal timing of urine collection or blood draw depends on the test ordered. For serum creatinine and blood urea nitrogen (BUN) levels, a random blood sample is appropriate. It is important to note that high protein ingestion can temporarily increase serum creatinine and urea levels. Hydration status can also significantly affect BUN results. Urine collection may involve a spot collection or collection over an extended period. A midstream urine sample is sufficient for a urinalysis. By collecting a midstream sample, the risk of contamination from epithelial cells or bacteria is lower. A 24-hour urine collection is used to assess creatinine clearance. Accurate collection is necessary for accurate results, which can make 24-hour urine collection difficult to conduct effectively outside the hospital setting. For this reason, an extended collection period, such as 5-8 hours, may be more practical (Gounden et al., 2024).

As the interpretation of GFR results has been previously reviewed, it is also necessary to understand some nuances about GFR values and how calculation methods affect results. GFR declines with age. A normal GFR for an adult male is 90 to 120 mL/min/1.73 m2, though by age 70, a healthy adult may have a GFR of 60 mL/min/1.73 m2 (Gounden et al., 2024). A measured glomerular filtration rate (mGFR) provides the most accurate picture of glomerular function; however, an accurate mGFR requires specialized testing with inulin infusion and serial blood draws to determine the inulin clearance rate. This test is not available in many

medical centers and is more time-consuming than other available calculation methods; therefore, other markers are often used to estimate GFR (eGFR), such as creatinine clearance and serum Cystatin C levels (Gounden et al., 2024).

Creatinine clearance can be used to assess the kidney's glomerular function. Creatinine is produced at a consistent rate in the body as a byproduct of muscle metabolism. Typically, creatinine is almost completely cleared from the blood by the kidneys. When creatinine is retained in the blood, it indicates compromised renal function (Gounden et al., 2024). Typically, a serum creatinine should be less than 1.2 for women and less than 1.4 in men. When the result exceeds these thresholds, it indicates kidney dysfunction (CDC, 2024). While creatinine clearance is used to estimate GFR, it tends to overestimate GFR by 10-20%, and kidney function may decline by 50% before serum creatinine becomes elevated (Gounden et al., 2024).

Urea, also known as blood urea nitrogen (BUN), is formed in the liver and is an end product of protein metabolism and the urea cycle. The kidneys eliminate most urea. When the kidneys are not functioning adequately, blood urea nitrogen becomes elevated. BUN can be elevated due to other causes, so it is important to note that this is not always an accurate assessment of renal function; however, when CKD is present, BUN will become elevated earlier in the disease process than creatinine (Gounden et al., 2024). A normal BUN typically ranges from 7-20, depending on age and comorbid conditions. Elevated BUN levels indicate renal dysfunction (CDC, 2024).

Cystatin C is a protein that is produced in every cell of the body at a constant rate. This protein is usually filtered out of the bloodstream in the glomerulus, but then reabsorbed and metabolized in the proximal renal tubules. As a result, there is typically not a significant amount of this protein in the excreted urine. Elevated serum Cystatin C levels indicate that the kidneys are not adequately filtering the

blood and that glomerular function is compromised. Some providers may prefer this test to estimate GFR because it does not vary with age, muscle mass, or diet, unlike serum creatinine levels. It also becomes elevated earlier than serum creatinine levels in the presence of CKD. In many equations used to estimate GFR, Cystatin C levels are used (Gounden et al., 2024).

As previously discussed, albuminuria can indicate glomerular dysfunction. However, multiple factors can lead to albuminuria. Therefore, CKD is considered when albuminuria is present on at least two occasions, at least 3 months apart. Protein in the urine can be measured using a 24-hour urine collection or a random sample. When a random sample is used, a urine-to-protein ratio is calculated. If a 24-hour collection is not feasible, first-morning urine is preferred for this test (Gounden et al., 2024). A urine albumin result of 30 mg/g or greater may indicate renal dysfunction. This test is often used for screening and diagnostic purposes, but is also used to monitor efficacy of treatment (CDC, 2024).

Tubular function tests can help the healthcare team determine how well the renal tubules are functioning in reabsorbing electrolytes and water and in regulating blood pH. Urine osmolality is a test that provides information regarding the body's ability to concentrate urine. For healthy, functioning kidneys, urine osmolality should be greater than 750 mOsm/kg H2O. Other conditions that affect urine osmolality, in addition to CKD, must also be considered (Gounden et al., 2024).

Urine analysis is a commonly used test that evaluates the characteristics of a urine sample through observation, chemical examination, and microscopic examination. Color is assessed: normal urine is straw-colored, and darker urine often indicates significant dehydration. Red or orange urine may result from blood or from the ingestion of certain foods and medications. Cloudy urine typically indicates a urinary tract infection. Specific gravity is included in the urine analysis and provides information about the kidneys' ability to concentrate urine. Normal urine

specific gravity is 1.003 to 1.030. It increases when urine is excessively concentrated and decreases when urine is excessively diluted. Urine analysis also provides information on the presence of protein, glucose, blood, ketones, bilirubin, urobilinogen, nitrite, and leukocyte esterase. Microscopic analysis of the urine can reveal red blood cells, which indicate glomerulonephritis, while white blood cells typically indicate pyelonephritis. Hyaline casts, fatty casts, and crystals in the urine can be observed in various conditions that affect kidney function (Gounden et al., 2024).

Section 5 Personal Reflection

Why are multiple tests used to diagnose and monitor CKD? Why might Cystatin C levels be preferred to calculate eGFR? What are the barriers to assessing mGFR? How can the timing or method of collection affect renal function test results?

Section 6: CKD Management

The first step in managing chronic kidney disease is identifying it. Evidence suggests that those at increased risk of developing CKD should be routinely screened with urinalysis, measurement of the urine albumin-to-creatinine ratio, serum creatinine, and eGFR (Vaidya & Aeddula, 2024). By identifying those at increased risk of CKD and offering blood and urine tests to detect the condition early, outcomes can be improved through delayed disease progression (CDC, 2025). Point-of-care urine tests are often used as a quick way to analyze urine at the bedside or in the clinic. This test cannot measure the amount of protein in the urine, but it can alert the healthcare team to its presence (CDC, 2024). CKD develops over time; therefore, serial lab results and a thorough history of symptoms can help the healthcare team diagnose CKD as early as possible (Vaidya & Aeddula, 2024).

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Physical assessment findings may not provide much information related to CKD in its early stages; however, some findings can indicate changes in kidney function. The healthcare worker should assess for changes in skin pigmentation and scratch marks from pruritus. Upon cardiac auscultation, a pericardial friction rub could indicate uremic pericarditis. Uremic frost occurs when high levels of BUN cause urea to be present in sweat, which dries to a fine, white powder on the skin. Hyperreflexia and muscle twitches may also be observed in early cases of CKD. Reports of vascular changes in the eye due to hypertension can provide valuable information about how increased blood pressure affects the microvasculature in other areas of the body, including the nephrons (Vaidya & Aeddula, 2024).

In addition to a complete medical history, review of symptoms, and a physical exam, other factors should be considered when determining a potential cause of alterations in renal function. The patient's medication list should be reviewed for medications that could be nephrotoxic. A social and environmental history, as well as a detailed family history, are important to obtain. In addition to laboratory tests previously discussed, imaging and tissue sample analysis may also be necessary. Ultrasound, intravenous urography, CT of the kidneys, ureter, and bladder, nuclear medicine studies, and MRI may all be used to assess kidney structure, including variations in size, shape, and symmetry, as well as evidence of obstruction, cystic disease, or reflux disease. Ultrasound-guided percutaneous kidney biopsy may be completed to study renal tissue. With specialized equipment, including light microscopy, an exact diagnosis may be determined (Stevens et al., 2024).

Information gathered through a complete assessment can also help to inform the healthcare team when planning treatment, assessing chronicity, assessing for genetic disease, and determining which treatments may be most successful. As previously discussed, serologic and urine tests are essential in evaluating for CKD. According to KDIGO practice guidelines, assessing for serum-free light chains, which are unbound proteins that are usually filtered out by the kidneys, is

becoming more clinically significant. Research advances have been made in interpreting the results and what they reveal about renal function. Genetic testing is increasingly being used as a diagnostic tool. Research suggests that genetic causes of CKD are more common than previously thought and may be a factor even in the absence of a significant family history of CKD (Stevens et al., 2024).

Treatment for CKD includes delaying progression of the disease through risk modification. Treatment guidelines recommend encouraging physical activity appropriate to the patient's health, tolerance, and ability. Specifically, 150 minutes of cumulative moderate-intensity activity is recommended for those who can do so. The duration and intensity should be adjusted as appropriate for each patient. For adults with stages G3-G5 CKD, the KDIGO guidelines recommend a consistent protein intake of 0.8 g/kg of body weight per day. Protein intake should not be restricted in children due to the negative effects this restriction could have on their growth and development. Older adults may require increased protein. The KDIGO guidelines suggest a restricted sodium intake of less than 2g per day. This means a suggestion of less than 5 mg of sodium chloride (table salt) per day. Blood pressure control is an important element of delaying CKD progression. The recommendation for hypertension management is to treat using medications and lifestyle changes for a target systolic blood pressure of less than 120 mmHg when tolerated. For those with diabetes, glycemic control can help reduce progression (Stevens et al., 2024).

Some causes of acute renal injury can be reversed, preventing the progression to CKD. Infections, nephrotoxic medications, hypotension, and hypovolemia must be identified and addressed to improve kidney function (Vaidya & Aeddula, 2024). Metabolic acidosis can lead to CKD progression. Bicarbonate supplementation for these patients has been found to delay progression. Scientific evidence suggests that smoking cessation slows the progression of CKD (Vaidya & Aeddula, 2024).

Certain medications may be used to treat CKD and to delay disease progression (Stevens et al., 2024). Medication dosages for individuals with CKD often require adjustment based on the patient's eGFR levels (Vaidya & Aeddula, 2024). KDIGO practice guidelines recommend prescribing a renin-angiotensin-system inhibitor (RASi), angiotensin-converting enzyme inhibitor (ACEi), or angiotensin II receptor blocker (ARB) for those with CKD who have severely increased albuminuria but do not have diabetes. These medications should be initiated earlier for patients with CKD and diabetes. While guidelines suggest that those with moderately increased albuminuria without diabetes could also benefit from these medications, those with moderately increased albuminuria and diabetes should start these medications immediately. Combinations of these medications are not recommended (Stevens et al., 2024).

Individuals who have CKD and type 2 diabetes or heart failure should be treated with a sodium-glucose cotransporter-2 inhibitor (SGLT2i), though it may need to be held during periods of fasting. Nonsteroidal mineralocorticoid receptor antagonists may be recommended for some patients, though serum potassium levels should be monitored. For individuals who have type 2 diabetes but have not been able to achieve glycemic control using metformin and SGLT2 inhibitors, glucagon-like peptide-1 receptor agonists (GLP-1 RA) medications are recommended. If an individual with CKD experiences gout, uric acid-lowering therapy should be offered, though it is not recommended for those with hyperuricemia who remain asymptomatic. Statin medications are recommended for those older than 50 with decreased eGFR for lipidemia management. Individuals with decreased eGFR who are not receiving dialysis treatment or who have not had a kidney transplant may be prescribed a statin/ezetimibe combination rather than a statin alone. Individuals ages 18-49 who are not receiving dialysis therapy and have not had a kidney transplant should be prescribed a statin if they also have known coronary artery disease, diabetes

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mellitus, prior ischemic stroke, or who have increased risk of a myocardial infarction according to an evidence-based risk assessment tool. Individuals with CKD and a history of ischemic cardiovascular disease should be prescribed low-dose aspirin (Stevens et al., 2024).

Medication management can be challenging for patients with CKD. In addition to multiple medications prescribed to prevent progression of CKD, the patient may require other medications for comorbid conditions. The healthcare team must be aware of how these other medications are metabolized in the body. For example, many antibiotics are excreted by the kidneys. If they are prescribed to someone with CKD, the medication can build up in the individual's body, leading to increased adverse effects and complications. Dosages of some medications may need to be changed to avoid nephrotoxicity. Medications commonly used to treat diabetes, such as metformin (Glucophage) and insulin, are excreted through the kidneys and can also accumulate in the body in patients with low GFR. Lower doses are often used, though some medications may be contraindicated once the GFR drops below a certain level. ACE inhibitors/ARBs, finerenone (Kerendia), spironolactone (Aldactone), or eplerenone (Inspra) are often used in patients with CKD to lower blood pressure and reduce albuminuria; however, these medications increase the risk for hyperkalemia in those with CKD. Diuretics, including medications like furosemide (Lasix) and torsemide (Demadex), are prescribed for those with CKD and/or heart failure. However, they can cause dehydration and hypokalemia in patients with a low GFR. Dosages of anticoagulants, such as warfarin (Coumadin) and others, must be carefully monitored in patients with low GFR, as reduced renal excretion can increase the risk of bleeding (National Kidney Foundation, 2025). Erythropoiesis-stimulating medications should be prescribed for those with anemia due to CKD (Vaidya & Aeddula, 2024).

Some over-the-counter medications, such as H2 blockers like famotidine (Pepcid) and cimetidine (Tagamet), and antacids, should be used with caution by those

with CKD. H2 blockers can accumulate in the body when GFR is low, and the appropriate dose should be confirmed with the provider before using these medications. Antacids contain calcium, magnesium, bismuth, aluminum, or a combination of these ingredients. All of these can accumulate in the body, and hypercalcemia, in particular, can adversely affect the cardiovascular system. The use of proton pump inhibitors, such as omeprazole (Prilosec OTC), esomeprazole (Nexium 24 hr), and others, should be monitored closely. Long-term use increases the risk of progression of CKD and can also contribute to worsened anemia or mineral and bone disorders in those with CKD. Certain bowel prep products used prior to colonoscopy are contraindicated with CKD, and safer alternatives must be used (National Kidney Foundation, 2025).

The healthcare team must be aware of certain risks associated with medications and CKD. Those with CKD are more susceptible to the nephrotoxic effects of medications. Renal function should be monitored closely in patients receiving medications with a narrow therapeutic window. It is also important that nurses and other members of the healthcare team know over-the-counter medications and herbal supplements that individuals with CKD use, as these can be harmful for these patients. Medications should be reviewed with the patient regularly to assess the accuracy of their medication list and to determine adherence to the treatment plan. If a patient is not adhering to their medication regimen, nurses should discuss this with the patient, as potential barriers may include access, cost, or adverse side effects that could be avoided with a different medication (Stevens et al., 2024). Patient education is critical to ensuring that patients and their caregivers understand what each medication is for and its importance. Collaborative relationships with other members of the healthcare team, especially pharmacists, can help optimize adherence to medication regimens and avoid complications that may result from complex regimens (Stevens et al., 2024).

Dietary counseling by a registered dietitian is recommended for many individuals with CKD. They may require guidance on very specialized diets, such as a low-potassium diet for those with hyperkalemia. Dietary counseling can also help ensure that the patient meets recommendations for sodium, protein, and water intake. Registered dietitians can help individuals troubleshoot barriers to implementing dietary changes and balance them with quality-of-life concerns. A plant-based Mediterranean-style diet and a low-fat diet are generally recommended for those with CKD to reduce cardiovascular risk (Stevens et al., 2024).

The healthcare team must be aware of the potential complications of CKD to avoid them or intervene early when they occur. Salt and fluid imbalances are common in patients with CKD. This may require a low-sodium diet and loop diuretics. Hypertension can also occur due to CKD. Close monitoring using frequent home blood pressure readings is necessary to determine if medications are being prescribed appropriately. Hyperkalemia is a potential complication that can occur, especially in patients experiencing oliguria and distal renal tubular dysfunction. Both metabolic changes in those with CKD, as well as certain medications often used to treat CKD, can contribute to hyperkalemia. Metabolic acidosis can occur in the late stages of CKD due to the retention of acidic compounds, potentially leading to osteopenia. Hyperphosphatemia is a complication of CKD that occurs when the kidneys fail to excrete phosphorus. This leads to hormone stimulation and hyperparathyroidism. Anemia is a common complication of CKD and should be monitored closely. Cardiovascular disease increases in severity as CKD progresses, so cardiac complications can occur when CKD is not well managed. Insulin resistance is associated with CKD and can contribute to the development of atherosclerosis. Malnutrition is a common complication of end-stage renal disease due to lack of appetite and inadequate protein intake. Uremic bleeding can occur due to impaired platelet function. Infections can be devastating for

those who have received a kidney transplant, and antirejection medications can increase the risk for malignancies. Monitoring medication regimens and effective communication among all members of the healthcare team are necessary to improve outcomes (Vaidya & Aeddula, 2024).

Once CKD has progressed to the point of renal failure (stages G4 and G5), renal replacement therapy (RRT) is necessary to replace the filtering function of the kidneys. This allows metabolic waste and excess fluid to be removed while maintaining electrolyte and acid-base balance. The two primary types of renal replacement therapy are dialysis and kidney transplant. Since CKD causes permanent damage to the kidneys, once kidney failure has been identified, dialysis is necessary for either the rest of the patient's life or until they receive a successful kidney transplant. A kidney transplant is the preferred option for renal replacement therapy; however, an organ for transplantation must be available, so dialysis may be necessary until transplant can occur (National Kidney Foundation, 2023c).

There are two types of dialysis. Hemodialysis uses a dialyzer to filter the blood as the kidneys would, then returns it to the body. This requires a vascular access site (National Kidney Foundation, 2023c). An autogenous arteriovenous fistula (AVF) is the preferred vascular access type for hemodialysis because it is the most durable and has the lowest complication rates. An AVF is created by a surgical procedure that joins an artery to a vein. As the vein adjusts to the increased arterial pressure, it dilates and thickens until it is suitable for use as a hemodialysis access site. Multiple anatomic locations are used to create an AVF. The radiocephalic configuration joins the radial artery to the cephalic vein at the wrist, creating access in the forearm. The brachiocephalic configuration joins the brachial artery to the cephalic vein at the antecubital fossa, with access in the upper arm. The brachiobasilic configuration joins the brachial artery and the basilic vein, though this procedure is more complex due to the basilic vein's location. Access for this

type of AVF is in the upper arm. Advanced planning for hemodialysis is important because once an AVF is created in surgery, it takes about six weeks before it can be used for dialysis access. Ideally, an AVF should be created 3-6 months before it is needed (Arasu et al., 2022).

An arteriovenous graft (AVG) is similar to an AVF but uses a polytetrafluoroethylene conduit that is tunneled to connect an artery and a vein. This procedure can use the prosthetic graft to connect the brachial artery to the cephalic or median cubital vein, creating access in the proximal forearm, or to connect the brachial artery to the axillary vein, creating access in the upper arm. An advantage of an AVG is that it can be used as soon as two weeks after placement, but AVGs are associated with a higher risk of infection and thrombosis compared to an AVF. Therefore, an AVG is typically only used when options for an AVF have been exhausted (Arasu et al., 2022).

A central venous catheter (CVC) can be used for patients who require hemodialysis but do not yet have a mature AVF or AVG, or who are awaiting an imminent kidney transplant. A CVC may also be used when all options for AVF or AVG access have been exhausted, the patient has severe cardiac disease, or life expectancy is short. While CVCs are much easier to place than other types of hemodialysis access, they have a higher infection rate, an increased risk of central venous stenosis, and poor long-term durability due to their propensity for thrombosis (Arasu et al., 2022).

When a patient has a hemodialysis vascular access site, the nurse must carefully assess the site to monitor for complications. Both arterial inflow and venous outflow should be assessed. To assess arterial outflow, the nurse should observe for signs of arterial insufficiency, such as pallor, limb atrophy, or tissue loss. The upper limb pulses should be palpable, and temperature of the extremity, capillary refill time, and sensorimotor response should be assessed. One way to assess

arterial inflow is to measure pulse oximetry in both upper extremities to determine whether there is a perfusion discrepancy. Venous outflow can be assessed by inspecting for signs of venous hypertension, including edema or prominent superficial collateral veins in the limb, face, or breast. Skin pigmentation or venous stasis ulcers can indicate severe venous outflow deficiency. A thrill should be palpated at the site of the outflow vein, indicating blood is flowing through the vessel. A continuous and low-pitched bruit should be heard upon auscultation of the site (Arasu et al., 2022).

Peritoneal dialysis utilizes the peritoneum as a natural filter to remove metabolic waste and excess fluid. In this procedure, a single-lumen silicone rubber catheter is surgically placed in the anterior abdominal wall. Hypertonic dialysate fluid is instilled into the abdominal cavity and left to dwell for a prescribed period. During this dwell period, solutes can diffuse from the blood into the dialysate via osmotic pressure across the peritoneal capillaries. Different glucose concentrations in the dialysate fluid are used to increase osmotic pressure and promote ultrafiltration. After the dwell period, the fluid is drained and replaced with another instillation of dialysate. The exact volume of dialysate used, fill time, drain time, glucose concentration, and number of cycles depend on several factors and are prescribed by the provider. This procedure can be performed manually or with a peritoneal dialysis machine, often called a cycler. It can be used continuously or intermittently, depending on the patient's needs. Nursing care of someone receiving peritoneal dialysis includes inspection and site care of the PD catheter insertion site, troubleshooting alarms from the cycler, such as drainage obstruction due to positioning, and knowledge regarding how to connect and disconnect from the cycler using evidence-based strategies to reduce the risk of contamination, which could lead to peritonitis (Teitelbaum, 2021).

Kidney transplantation from a living or deceased donor is the preferred treatment for kidney failure, as it is associated with better quality of life, lower mortality,

fewer dietary restrictions, and lower treatment cost compared to dialysis. Ideally, patients receive a kidney transplant before dialysis is necessary. Some conditions may prevent a patient from being eligible to receive a kidney transplant. These include advanced age, severe cardiovascular disease, active or recent cancer, dementia or poorly controlled mental illness, alcohol or drug misuse, or any barrier that would prevent the patient from adhering to a strict medication regimen that is necessary to prevent organ rejection (Mayo Clinic Staff, 2025).

Certain populations may require special considerations when managing chronic kidney disease. The treatment approach for kidney disease in an otherwise healthy 30-year-old will differ from that of an 85-year-old with comorbid conditions or a 3-year-old with congenital renal abnormalities. In children, it is estimated that 40-50% of chronic kidney disease is due to the presence of a congenital anomaly of the kidneys or urinary tract. These children typically experience a slower disease progression and a higher likelihood of polyuria. In the care of children with CKD, the healthcare team must be diligent about including caregivers in education and decision-making. Caregivers must understand their child's condition to help improve outcomes. As the child ages into adolescence and young adulthood, the healthcare team will need to allow appropriate independence in managing care while providing support as the child transitions to adult healthcare. Physical growth and development are a significant aspect of CKD management in children and adolescents. Normal physiologic processes, such as puberty, can contribute to disease progression due to the period of rapid growth and increase in muscle bulk. In very young children, it is essential to note that, although nephron formation is complete at 36 weeks' gestation, nephrons continue to grow and mature rapidly in the first year of life, with growth continuing into the second year, and maturation can continue up to 4 years of age. As a result, GFR increases during these early developmental years. Children with CKD may exhibit this increase in GFR, experience a period of stability, and then

experience a decline in renal function as they progress into adolescence and adulthood. Since children and adolescents should have optimally functioning kidneys, an eGFR of less than 90 ml/min/1.73 m2 is considered indicative of decreased kidney function. Neurodevelopment is also a special focus when managing CKD in this population. CKD can affect a child's physical and cognitive development, with lasting effects on education and future employment. Individualized care is utilized to optimize development for these children (Stevens et al., 2024).

Older adults must also be given special consideration when managing CKD. Individuals in this population, especially those over age 75, often have multiple comorbidities, polypharmacy, frailty, cognitive impairment, and gerontopsychiatric disorders. The prevalence of these conditions is increased for those with CKD. The patient's advanced age must be considered when interpreting lab results. An eGFR based on serum creatinine will overestimate GFR in older adults with sarcopenia, the age-related loss of muscle mass. This may result in medications prescribed at doses that are too high for the individual. Urine-to-creatinine ratio results may be misleading as well. The result may be falsely high due to the lower creatinine quantity in the denominator of the ratio. The usual interventions prescribed to slow the progression of CKD in younger adults may not be practical or even safe for older adults. Implementing strategies to lower blood pressure may lead to dizziness, falls, and fractures among older adults. Multiple comorbidities require complex care and multidisciplinary communication. Treatment goals may also differ from those of younger adults due to the inherent shortened life expectancy in advanced age. Quality of life may become a much more significant factor in making healthcare decisions than years of survival. Clear and open communication among the healthcare team, the patient, and caregivers is essential to ensure that the patient and their family can make informed decisions. Due to cognitive decline in some patients, conversations may need to be

repeated. The older adult population is the largest cohort with CKD; therefore, awareness of this population's needs is necessary to individualize care and promote optimal outcomes (Stevens et al., 2024).

Sex and gender-related factors can influence the progression of CKD, as well as disease presentation, pathophysiology, response to treatments, complications, and outcomes. Sex refers to an individual's biological attributes, and sex-based variations in genetics, physiology, immunology, and anatomy affect renal function. Gender refers to sociocultural factors and may include identities, roles, and societal views of the attributes of males and females. Some medications used to treat CKD are not safe to use during pregnancy; therefore, contraceptive counseling or consideration of safe alternative medications may be necessary for female patients. CKD can negatively impact fertility in both males and females, and pregnancy is a significant risk factor for CKD progression and complications, as well as adverse outcomes for the mother and infant. Hormones and hormone changes can affect pharmacokinetics. Women are more likely to report adverse effects of ACE inhibitors. Though the reasons are unclear, research has found differences in the detection, recognition, monitoring, referral, and management of CKD between men and women. Healthcare workers should be aware that sex and gender factors may impact those who identify as transgender, gender-diverse, or non-binary differently, as biological sex and hormone therapies may also impact disease progression and management. Understanding methods to foster transgender cultural safety in the healthcare setting can improve outcomes for patients, as they are more likely to seek healthcare and continue with follow-up care when these unique needs are met (Stevens et al., 2024).

Section 6 Personal Reflection

What assessment information is necessary for the nurse to obtain when CKD is suspected? What is the primary focus of CKD treatment? What non-pharmaceutical strategies are used to manage CKD? What types of medications might be used to manage CKD? Why do you think polypharmacy is especially dangerous for those with CKD? What factors must be considered for special populations?

Section 7: Nursing Implications

Caring for patients with CKD requires an interdisciplinary team approach. Nurses, physicians, dietitians, pharmacists, psychologists, and patient educators can all help facilitate this by contributing to effective communication and advocating for the patient.

The primary goals of nursing care for patients with CKD are to prevent or slow disease progression, promote physical and psychosocial well-being, and monitor disease and treatment-related complications. The best way to prevent the progression of CKD is to identify the risk factors for progression and then to collaborate with the patient and their caregivers to focus on modifiable risk factors. For patients with diabetes and/or hypertension, nursing care should focus on helping patients understand strategies to maintain their target ranges for blood glucose and blood pressure. Lifestyle changes and medication adherence are both necessary to reduce the risk of progression of CKD due to these conditions (Vaidya & Aeddula, 2024).

Nurses should provide individualized patient education to help patients understand and manage their condition and promote overall well-being. Patient and caregiver education should include an overview of CKD, treatment, and self-

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management techniques. The nurse will need to assess educational needs to determine baseline knowledge, CKD stage, and current and future treatment plans. The nurse must evaluate the effectiveness of patient education and be sensitive to the patient's psychological needs.

An effective way to evaluate the patient and caregiver's knowledge of CKD is to ask questions such as "What do you know about your condition?", "What would you like to know about chronic kidney disease?", and "What would you like to know about treating your illness?" These open-ended questions can provide valuable information about the patient's understanding of their condition and specific concerns.

Patient education for CKD is ongoing. In the early stages of CKD, the nurse should focus on encouraging self-management and interventions the patient can implement, such as dietary changes and glycemic control, to slow disease progression. As CKD advances to later stages, patients and their caregivers will need education regarding renal replacement therapy and transplant. There are many options available for RRT, which may be overwhelming for the patient and the caregiver to consider. When introduced early, the patient and caregiver can process potential options before they become necessary to initiate. The nurse can ask questions to help the patient think towards the future, such as "Have you thought about what treatments you may or may not want in the future?"

Self-management strategies for CKD usually require weight management. Nurses will need to discuss healthy eating and activity habits. Some patients will require specialized diets and may need further education by a registered dietitian. Any prescribed dietary restrictions should be reviewed with the patient and caregiver, and especially the person who prepares the patient's meals, to verify understanding of which foods to eat and which to avoid. Typically, the

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recommended diet for those with CKD includes foods that are fresh, lean, low-fat, low-sugar, and low-cholesterol. Avoiding alcohol and nephrotoxins can also help to slow progression. Patient education should focus on smoking cessation if that is an issue for the patient. Nurses should also educate the patient on protecting themselves from injury and infection, including strategies such as diligent handwashing to prevent illness, avoiding contact with family and friends who are ill, and identifying and removing fall risks in the home, such as area rugs. The nurse should review the signs and symptoms of infection with the patient and caregiver and instruct them regarding when to contact the provider (Stevens et al., 2024).

Prior to initiating any patient education, the nurse should assess the patient and/or caregiver's ability to self-manage their medical care. By asking open-ended questions, such as "How is your physical health?", "How is your emotional health?", "What are your priorities?", "What would improve your quality of life?", the nurse can individualize education to address the patient's concerns and priorities. Based on the patient's responses, the nurse can also help the patient and caregiver to improve self-management skills.

Nurses should remember that patients living with CKD are at increased risk for psychological challenges, including anxiety, depression, and stress. They may feel vulnerable or self-conscious because of a dialysis access site. They may feel anxious about decisions that must be made. Those who require a kidney transplant may also feel guilty about accepting a deceased donor's organ. Polypharmacy, dietary and activity restrictions, appointments, treatments, and the effects of these things on the patient's employment may lead to them feeling overwhelmed, or as if their condition is a burden to their loved ones. Nurses can help improve psychological outcomes related to CKD by assessing the patient's psychosocial status and coping mechanisms, and then helping them identify

strategies, resources, and interventions to support their mental health (Stevens et al., 2024).

Nurses must monitor for the many complications that can result from CKD and its treatments. A thorough nursing assessment will aid in identifying signs and symptoms that may indicate a problem and provide information on any changes that have occurred since the last assessment. The physical assessment and vital signs can also inform the healthcare team regarding the effectiveness of treatment. A nursing assessment for a patient with CKD must include vital signs including pulse oximetry, a pain assessment, evaluation of intake and output, weight, mental status, energy level, reflexes, skin color and integrity, heart and lung sounds, whether blood has been observed in the stools or sputum, psychological status, and the patient's ability to perform activities of daily living (Vaidya & Aeddula, 2024).

There are specific nursing considerations for patients who are in the late stages of CKD. The dialysis access site must be assessed, and any issues, including occlusion or signs of infection, must be reported to the provider. Access sites should be kept clean and dry. The extremity that is used for dialysis access must not be used for any other procedures. This may require an identification band to alert other members of the healthcare team to this restriction. It is especially important for patients receiving RRT for the nurse to report any signs and symptoms of complications to the provider. These may include changes in vital signs, nausea, vomiting, chest or back pain, abdominal cramping, fever, or chills. The access site should be assessed for signs of infection, including redness, warmth, tenderness, purulent discharge, sores, or swelling. For patients with a hemodialysis arteriovenous graft or fistula, the nurse should assess the access point for patency by palpating for a thrill (a vibration at the access point) or auscultating for a bruit (a swishing sound at the access point). Any changes in sensation, color, temperature, or appearance of the site should be reported immediately. Blebs,

ballooning, and bulging are all abnormalities that should not be present at the access site. Patients who require a kidney transplant must receive extensive pre- and post-transplant care and support (Mayo Clinic Staff, 2025).

Nurses must also remember that patients in end-stage renal disease may refuse treatment or choose to pursue palliative care. By providing education, nurses empower patients and their caregivers to make decisions that are best for them. Nurses can collaborate with other members of the healthcare team to ensure the team understands the patient's and their caregivers' decisions and priorities (Stevens et al., 2024).

Section 7 Personal Reflection

What are the primary goals of CKD nursing care? What should patient education focus on during the early stages of CKD? How can a patient with CKD make lifestyle changes to prevent progression of CKD? Why do you think patients with CKD are at risk for psychological challenges? What considerations must be made for patients in the late stages of CKD?

Section 8: Case Study

Mrs. Andrews, a 70-year-old black female, presents to the primary care clinic for a routine physical exam. She has a history of type 2 diabetes mellitus, hypertension, obesity, GERD, and osteoporosis. Today she has no specific complaints, though she continues to feel tired frequently and reports that her feet sometimes swell. Mrs. Andrews is prescribed medications to manage her medical conditions, though she reports she is not always diligent about checking her blood sugar each day.

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The provider orders lab testing to assess renal function. Mrs. Andrews later asks the nurse why the doctor wants to check her kidneys. What is the most appropriate response to give Mrs. Andrews?

a. Everyone aged 65 and older is ordered this type of testing
b. She has multiple risk factors that could cause kidney problems, so it is best to monitor for them and intervene early if there are issues
c. Continued fatigue indicates that kidney function should be assessed
d. Non-compliance with blood glucose monitoring requires additional lab tests

The lab results are as follows:

Serum creatinine: 1.5 mg/dL
BUN: 22 mg/dL
eGFR: 70 mL/min/1.73m2
Albumin-to-Creatinine Ratio: 140 mg/g

What information is needed when interpreting these lab results? Select all that apply.

a. Time of day the sample was collected
b. What medications the patient is taking
c. Pulse oximetry measurement
d. Last measured blood pressure
e. Previous renal function test results
f. Lipid panel results

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The nurse reviews previous lab results from Mrs. Andrews's appointment four months ago. Today's results are consistent with those from the previous test. What can be inferred about these results?

a. Mrs. Andrews's kidneys are functioning normally
b. Mrs. Andrews may have some renal dysfunction
c. Mrs. Andrews has advanced chronic kidney disease
d. Mrs. Andrews is likely dehydrated

Based on the lab results, how would you classify Mrs. Andrews's diagnosis of CKD?

a. Metabolic kidney disease, G3, A2
b. Acute kidney injury, G1, A1
c. Diabetic and hypertensive CKD, G2, A2
d. Osteopenic CKD, G2, A1

What patient education is necessary for Mrs. Andrews today? Select all that apply.

a. Information about CKD
b. Strategies to meet blood glucose goals
c. Strategies to increase fluid intake
d. Strategies to maintain target blood pressure
e. Medication adherence
f. Lifestyle changes that can reduce risk of progression
g. Information about kidney transplants
h. Education about how diabetes and hypertension affect the kidneys

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What specific changes can Mrs. Andrews make to reduce CKD progression?

a. Decrease physical activity, increase fluid intake, check blood glucose as ordered

b. Increase physical activity, make dietary changes to promote weight loss, adhere to medication regimen

c. Increase physical activity, increase sodium intake, Decrease potassium intake

d. Decrease physical activity, increase protein intake, make dietary changes to promote weight loss

What factors about Mrs. Andrews's medical history contribute to increased risk of CKD progression? Select all that apply.

a. Age

b. Race

c. Sex

d. Type 2 diabetes

e. Hypertension

f. GERD

g. Osteoporosis

h. Obesity

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Section 9: Conclusion

Chronic kidney disease is a highly complex condition that affects multiple organ systems. The complexity and interrelationships among conditions that lead to CKD can easily confuse patients, caregivers, and healthcare workers who are not familiar with CKD. This can lead to poor understanding of the disease, medication errors and non-adherence, and progression of the disease. When nurses understand how the kidneys normally function, are knowledgeable about what CKD is, understand how to interpret renal function lab results, and are skilled at identifying the early signs and symptoms of the disease, its risk factors, and its effects on the cardiovascular system, CKD interventions can be implemented early. Nurses who understand the treatment goals and strategies at different stages of CKD are prepared to optimize outcomes through effective nursing interventions and patient education. Though CKD is a widely prevalent condition with serious implications, nurses can improve individual outcomes through evidence-based knowledge and clinical practice.

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