Making Sense of CKD

A Concise Guide for Managing Chronic Kidney Disease in the Primary Care Setting

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NKDEP National Kidney Disease Education Program

A program of the National Institutes of Health

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I. About this Guide

Primary care professionals are critical to Chronic Kidney Disease (CKD) care. Many CKD care issues overlap with those of diabetes and hypertension, and addressing CKD early—prior to nephrology referral—can improve patient outcomes. However, the numerous, sometimes conflicting guidelines for CKD can make understanding and providing appropriate care challenging. This may be especially true for busy primary care professionals who are charged with managing a broad spectrum of conditions.

Developed by the National Kidney Disease Education Program (NKDEP), this guide is intended to help busy health care professionals manage adult CKD patients in the primary care setting. The guide emphasizes the most important considerations for evaluating and managing CKD patients, including identifying and slowing progression among patients at highest risk for progression to kidney failure.

In addition, this guide highlights a variety of resources — including patient education materials, clinical tools, and professional reference materials — to help providers assess, manage, and educate patients with CKD in the primary care setting.

Addressing CKD early can improve patient outcomes.

II. About CKD

The kidneys regulate the composition and volume of blood, remove metabolic wastes in the urine, and help control the acid/base balance in the body. They produce erythropoietin needed for red-blood cell synthesis and activate vitamin D needed for calcium absorption and bone health.

CKD is detected and monitored by two tests:

- estimated glomerular filtration rate (eGFR)
- urine albumin-to-creatinine ratio (UACR)

CKD is typically a progressive disease. It is defined as

- reduction of kidney function — defined as an eGFR < 60 mL/min/1.73 m^2 for > 3 months

AND/OR

- evidence of kidney damage, including persistent albuminuria — defined as ≥ 30 mg of urine albumin per gram of urine creatinine for > 3 months

Kidney failure is typically defined as an eGFR < 15 mL/min/1.73 m^2.

The key issues in managing CKD are

- ensuring the etiology is correct
- implementing appropriate therapy
- monitoring the patient
- screening for CKD complications
- educating the patient

Not all patients with decreased eGFR or low grade albuminuria will progress to kidney failure. It is important to identify and slow progression among patients at high risk for progressive disease. In general disease progression is often associated with

- high levels of albuminuria,
- progressive decrease in eGFR, and
- poorly controlled blood pressure.

However, imprecision of biomarkers for kidney function and damage, as well as variability in disease progression between individuals, suggests that progression risk must incorporate a variety of clinical characteristics including eGFR, UACR, rate of change, and blood pressure. Until validated algorithms are available, clinicians are cautioned about predicting prognosis based on any single measurement of a particular biomarker.

Not all patients with decreased eGFR or low grade albuminuria will progress to kidney failure. It is important to identify and slow progression among patients at high risk for progressive disease.

The key components to slowing progression of CKD are to

- control blood pressure (with angiotensin-converting enzyme inhibitors, angiotensin receptor blockers)
- reduce albuminuria
- manage diabetes
- avoid acute kidney injury

As eGFR declines, complications occur more commonly and are more severe. These may include

- cardiovascular disease (CVD) and dyslipidemia
- anemia due to impaired erythropoiesis and low iron stores
- mineral imbalance and bone disorder (calcium, phosphorus, and vitamin D)
- hyperkalemia
- metabolic acidosis
- malnutrition (low serum albumin)
- fluid and salt retention, often associated with accelerated hypertension

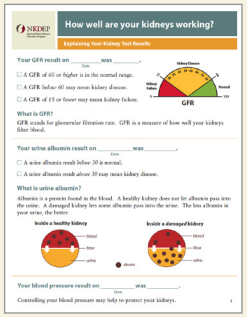
Informing a patient about any chronic disease is challenging. Accepting the diagnosis may be difficult. Most people with CKD have no symptoms until the eGFR is significantly reduced. Education and early preparation for the transition to kidney failure may be beneficial.

CKD Risk Factors

- Diabetes
- Hypertension
- Family history of kidney failure
- Cardiovascular disease
- HIV infection
- Immunological diseases

III. Identifying and Evaluating CKD

TEST AND ITS RELEVANCE	RESULTS	ASSESSMENT
Estimated Glomerular Filtration Rate (eGFR) *Estimates kidney function* As eGFR declines, complications are more likely and more severe.	**eGFR (mL/min/1.73m²)** Not diagnostic of CKD ≥ 60 CKD 15 – 59 Kidney failure < 15	• Track eGFR over time to monitor effectiveness of therapy. • Stable eGFR may indicate therapy is working. • Decline of eGFR may reflect progression of CKD.



RELEVANT RESOURCE

Explaining Your Kidney Test Results: A Tear-off Pad for Clinical Use includes 50 easy-to-read patient education sheets for explaining urine albumin and GFR results to patients. nkdep.nih.gov/resources/explaining-kidney-test-results.shtml

Additional Information

About eGFR

- eGFR reflects the total filtration by all functioning nephrons.
- As nephrons are damaged or destroyed, eGFR declines.
- As eGFR declines
  - The volume of urine may not change significantly, but blood composition changes.
  - Monitoring laboratory data may identify CKD complications.

Estimating GFR

- Most laboratories routinely report eGFR with all serum creatinine determinations in adults.
- If your lab does not report eGFR, it can be calculated:
  - NKDEP offers calculators online and as downloadable applications for estimating GFR. Visit: nkdep.nih.gov/gfr-calculator.
  - Serum creatinine level, age, gender, and race must be entered.

Limitations of eGFR

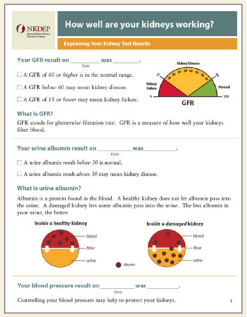
- eGFR provides an **estimate** of kidney function.
- Current estimating equations have only an 80 to 90% chance of being within +/-30% of the measured GFR.
- This uncertainty increases significantly for eGFRs above 60.

III. Identifying and Evaluating CKD, *(cont.)*

TEST AND ITS RELEVANCE	RESULTS	ASSESSMENT
Urine Albumin-to-Creatinine Ratio (UACR) *Reflects kidney damage* • preferred measure for screening, assessing, and monitoring kidney damage • unaffected by variation in urine concentration (unlike a dipstick test for urine albumin) • result (mg/g) approximates milligrams of albumin excreted in 24 hours • may be the earliest sign of glomerular diseases including diabetic kidney disease Other common names include • microalbumin • urine albumin • albumin-to-creatinine ratio (ACR) • microalbumin/ creatinine ratio	**UACR (mg albumin/ g creatinine)** Normal 0 – 29 Albuminuria ≥ 30	• Evaluate UACR over time to assess response to therapy and monitor progression of CKD. • Elevated albuminuria may reflect higher risk for progression. • A decrease in albuminuria may reflect response to therapy and may be associated with improved renal and cardiovascular outcomes. **Additional Information** **About UACR** • Normally functioning kidneys excrete very small amounts of albumin in the urine. • Albuminuria – usually reflects damage to the glomerulus — the "filter" of the nephron – is an independent risk factor for CKD progression – is considered a marker for CVD and mortality in hypertension • Reducing urine albumin to normal or near-normal levels may improve cardiovascular prognosis. **Increases in UACR** • Urine albumin may be transiently increased – after strenuous exercise – with fever or infection • Urine albumin may be elevated with – dehydration – hyperglycemia – congestive heart failure

III. Identifying and Evaluating CKD, *(cont.)*

UACR, *(cont.)*



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Limitations of UACR

- Urine albumin measurement is not standardized. Common assays have shown significant imprecision, varying by 40% across albumin concentrations.
- Daily variation in albumin excretion within individuals may confound interpretation and risk assessment.
- Albuminuria levels are affected by glycemia, blood pressure, and type of anti-hypertensive medication.

Once CKD is identified, further evaluation may establish etiology and provide baseline data for both the primary care provider and nephrologist, when consultation is needed.

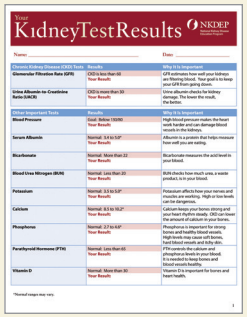
Initial evaluation may include

- glucose (A1C/eAG)
- creatinine with eGFR
- blood urea nitrogen
- electrolytes
- albumin
- calcium
- phosphorus
- fasting lipid panel
- complete blood count
- complete urinalysis
- renal ultrasound
- dilated retinal exam

Further work-up may include

- Tests of auto-immunity
  - antinuclear antibody test (ANA)
  - rheumatoid factor (RF)
  - complement 3 (C3)
  - complement 4 (C4)
  - ANCA
- Paraprotein assessment in adults over age 40
  - serum protein electrophoresis (SPEP)
  - urine protein electrophoresis (UPEP)
- Hepatitis Serologies
  - hepatitis B serology (HBsAg)
  - hepatitis C serology (antiHCV)

Further evaluation, *(cont.)*


Your KidneyTestResults NKDEP

Name: _____ Date: _____

Chronic Kidney Disease (CKD) Tests	Results	Why It Is Important
Glomerular Filtration Rate (GFR)	CKD is less than 60 Your Results	GFR estimates how well your kidneys are filtering blood. Your goal is to keep your GFR from going down.
Urine Albumin-to-Creatinine Ratio (UACR)	CKD is more than 30 Your Results	Urine UACR checks for kidney damage. The lower the result, the better.
Other Important Tests	**Results**	**Why It Is Important**
Blood Pressure	Goal: Below 130/80 Your Results	High blood pressure makes the heart work harder and can damage blood vessels in the kidneys.
Serum Albumin	Normal: 3.4 to 5.0* Your Results	Albumin is a protein that helps measure how well you are eating.
Bicarbonate	Normal: More than 22 Your Results	Bicarbonate measures the acid level in your blood.
Blood Urea Nitrogen (BUN)	Normal: Less than 20 Your Results	BUN checks how much urea, a waste product, is in your blood.
Potassium	Normal: 3.5 to 5.0* Your Results	Potassium affects how your nerves and muscles function. High or low levels can be dangerous.
Calcium	Normal: 8.5 to 10.2* Your Results	Calcium keeps your bones strong and your heart rhythm steady. CKD can lower the amount of calcium in your bones.
Phosphorus	Normal: 2.7 to 4.6* Your Results	Phosphorus is important for strong bones and healthy blood vessels. CKD can cause high levels, leading to hard blood vessels and itchy skin.
Parathyroid Hormone (PTH)	Normal: Less than 65 Your Results	PTH controls the calcium and phosphorus in your blood. It is needed to keep bones and blood vessels healthy.
Vitamin D	Normal: More than 30 Your Results	Vitamin D is important for bones and heart health.

*Normal ranges may vary.

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RELEVANT RESOURCE

Your Kidney Test Results helps providers assess and discuss test results with CKD patients. nkdep.nih.gov/resources/kidney-test-results.shtml

Determining Etiology

- If a patient with diabetes has retinopathy, albuminuria, and negative serologic work-up (above), it is reasonable to assume the diagnosis is diabetic kidney disease.
- Patients who do not conform to diabetic kidney disease criteria should be discussed with a nephrologist. (See Collaborate with Nephrologist on page 20.)

Additional laboratory data may include

- iron studies when anemia is present (see Anemia on page 12)
- vitamin D and intact parathyroid hormone (iPTH) (see Mineral and Bone Disorders on page 13)

IV. Slowing Progression

THERAPEUTIC GOAL AND ITS RELEVANCE	RANGES/GOALS	INTERVENTIONS
Control Blood Pressure Blood pressure control slows progression of CKD and lowers CVD risk. Multiple antihypertensive medications may be required to control blood pressure.	< 140/90 mmHg	• Renin angiotensin aldosterone system (RAAS) antagonists — i.e., angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) — are often used to – control blood pressure – delay progression – reduce albuminuria – protect against heart disease • Monitor serum potassium in patients on RAAS antagonists: – RAAS antagonists increase the risk for hyperkalemia. – Initiate dietary potassium restriction if necessary to maintain potassium <5 mEq/L. • Diuretics are prescribed to treat fluid overload and high blood pressure, and may help control serum potassium levels. **Additional Information** • Consider an ARB if patient experiences persistent non-productive cough with use of ACEi. • Concurrent use of ACEi and ARB is not indicated. • Sodium restriction enhances efficacy of RAAS antagonists.
Reduce Albuminuria Elevated protein levels within kidney tubules may exacerbate kidney damage. Decreased albuminuria is associated with slower CKD progression, particularly in people with diabetes.	Reduce or stabilize the amount of albumin lost in the urine. (See UACR on page 5.)	• RAAS antagonists are associated with a reduction in albuminuria. • Tobacco cessation may lower albuminuria. **Additional Information** Observational data suggests weight loss and sodium restriction may lower albuminuria.

IV. Slowing Progression, *(cont.)*

THERAPEUTIC GOAL AND ITS RELEVANCE	RANGES/GOALS	INTERVENTIONS
Manage Diabetes Good control of newly diagnosed diabetes may delay the onset and/or slow progression of CKD. Tight control in diabetes of long duration may not slow CKD progression.	Individualize A1C/ eAG goal eAG = estimated average glucose	• Dose adjustment of medications may be needed in CKD. As eGFR declines, renal metabolism of insulin and certain oral diabetes medications is reduced, potentially causing hypoglycemia. • Unexplained improvement in glucose control may reflect progression of CKD. • Consider less-stringent control for – patients with histories of hypoglycemia – elderly patients – patients with multiple co-morbid conditions **Additional Information** • Continue to manage other diabetes-related complications (e.g., retinopathy, neuropathy). • If you refer your CKD patient to a nephrologist, reporting the diabetes status may be helpful.

THERAPEUTIC GOAL AND ITS RELEVANCE

Manage Diet

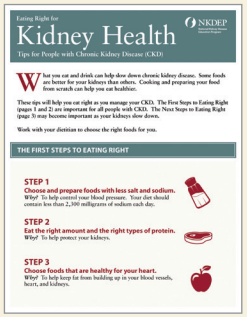
Refer to a registered dietitian (RD) for Medical Nutrition Therapy as needed. To find a registered dietitian, visit eatright.org/programs/rdfinder.

As time allows and based on patient interest, discuss basic steps:

- reducing sodium intake
- reducing protein intake, if excessive (see Metabolic Acidosis on page 17)
- substituting liquid oil in place of solid or hydrogenated fats
- limiting phosphorus, including added phosphorus (see Phosphorus on page 15)
- limiting potassium when serum level is elevated (see Hyperkalemia on page 17)

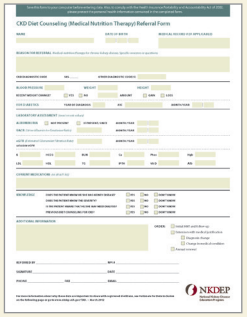
To treat hypoglycemia in diabetics with hyperkalemia, use glucose tablets, cranberry juice cocktail, or apple juice instead of orange juice or cola.

ADDITIONAL INFORMATION AND RESOURCES


Eating Right for
Kidney Health
Tips for People with Chronic Kidney Disease (CKD)
NKDEP
What you eat and drink can help slow down chronic kidney disease. Some foods are better for your kidneys than others. Cooking and preparing your food from scratch can help you eat healthier.
These tips will help you eat right as you manage your CKD. The First Steps to Eating Right (pages 1 and 2) are important for all people with CKD. The Next Steps to Eating Right (page 3) may become important as your kidneys slow down.
Work with your dietitian to choose the right foods for you.
THE FIRST STEPS TO EATING RIGHT
STEP 1
Choose and prepare foods with less salt and sodium.
Why? To help control your blood pressure. Your diet should contain less than 2,300 milligrams of sodium each day.
STEP 2
Eat the right amount and the right types of protein.
Why? To help protect your kidneys.
STEP 3
Choose foods that are healthy for your heart.
Why? To help keep fat from building up in your blood vessels, heart, and kidneys.

RELEVANT RESOURCES

Eating Right for Kidney Health helps patients make sense of the multiple, complex diet recommendations for people living with CKD: nkdep.nih.gov/resources/eating-right.shtml


CKD Diet Counseling (Medical Nutrition Therapy) Referral Form
NKDEP

CKD Diet Counseling (Medical Nutrition Therapy) Referral Form makes it easy to share important patient data with the consulting dietitian: nkdep.nih.gov/mnt-referral

THERAPEUTIC GOAL AND ITS RELEVANCE

Avoid Acute Kidney Injury (AKI)

CKD patients have increased susceptibility to nephrotoxic agents and are at high risk for AKI. AKI may accelerate CKD progression.

To lower risk for AKI, use caution with nephrotoxic medications/treatments that may exacerbate kidney damage:

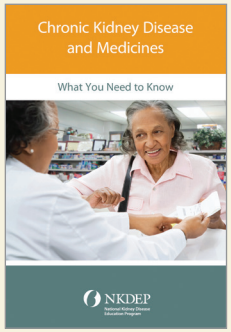
- Avoid non-steroidal anti-inflammatory drugs (NSAIDs).
  - Do not prescribe NSAIDs.
  - Counsel patients to avoid over-the-counter NSAIDs.
  - Common NSAIDs include diflunisal, celecoxib, ibuprofen, and naproxen.
- Avoid other potentially nephrotoxic drugs (e.g., quinolones, beta lactams, sulfonamides).
- Use caution with intravascular administration of iodinated contrast agents. Consider alternative imaging techniques that do not require iodinated contrast media.

ADDITIONAL INFORMATION AND RESOURCES

Acute Kidney Injury Diagnosis/Characterization

- A rapid loss of kidney function defined by either
  - an absolute increase in serum creatinine of ≥ 0.3 mg/dl

    OR
  - a percentage increase in serum creatinine of ≥ 50%
- Characterized by
  - accumulation of nitrogenous wastes (uremic toxins)
  - edema
  - disruption of electrolyte (e.g., potassium) or acid-base balance
- Early symptoms may include
  - fatigue
  - reduced urine output
  - edema



RELEVANT RESOURCE

CKD and Medicines: What You Need to Know provides an overview of medicines for people with CKD and encourages patients to turn to pharmacists for information and support: nkdep.nih.gov/resources/ckd-medicines.shtml

V. Preventing, Monitoring, and Treating Complications

Many of the recommendations for CKD are based on findings from studies of dialysis patients.

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION
Cardiovascular Disease (CVD) Patients with CKD are at high risk for developing CVD; the risk increases as eGFR declines. CVD is the leading cause of mortality in CKD.	LDL cholesterol < 100 mg/dL	• Controlling dyslipidemia is beneficial in people with CKD. • Aspirin therapy is recommended unless otherwise contraindicated. **Additional Information** Nontraditional risk factors for CVD in CKD include • anemia • albuminuria • vascular calcification secondary to mineral imbalance
Anemia Anemia may develop early during the course of CKD due to inadequate synthesis of erythropoietin by the kidneys. Hemoglobin is used to assess anemia in CKD. More frequent monitoring may be necessary based on severity of anemia and as eGFR declines. Uncomplicated anemia of CKD is usually normocytic and normochromic.	Normal Hemoglobin: 11 – 12 g/dL **Without CKD** Women: 12 – 16 g/dL Men: 14 – 17 g/dL Transferrin Saturation (TSAT) > 20% Ferritin > 100 ng/mL **Without CKD** Women: 18 – 160 ng/mL Men: 18 – 270 ng/mL	• Further evaluation may identify correctable causes of anemia (e.g., GI blood loss) including iron deficiency. Recommended for evaluation – iron studies which include serum ferritin and transferrin saturation (TSAT) results – peripheral blood smear may be indicated depending on peripheral blood smear results and iron levels – stool for blood – serum folate level – vitamin B12 • Oral iron supplements may correct mild iron deficiency in CKD. Intravenous (IV) iron may be considered if oral iron is inadequate or not well tolerated. • Injectable erythropoiesis-stimulating agents (ESAs) are commonly used to correct anemia in patients on dialysis.

V. Preventing, Monitoring, and Treating Complications, *(cont.)*

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION
Anemia, *(cont.)*		**Erythropoiesis-Stimulating Agents in CKD** • Recent studies have not shown significant benefit from ESAs and have even suggested they may cause harm. • For patients not on dialysis, the Food and Drug Administration recommends providers consider starting ESA therapy only when – hemoglobin is less than 10 g/dL **AND** – rate of hemoglobin decline shows the patient may require a red blood cell (RBC) transfusion **AND** – reducing the risk of alloimmunization and/or other RBC transfusion-related risk is a goal. • Collaborate with a nephrologist when considering ESAs. (See Collaborate with Nephrologist on page 20.)
Mineral and Bone Disorder (MBD) Mineral imbalance may • develop due to changes in levels of vitamin D and parathyroid hormone • result in abnormal serum calcium and phosphorus levels and renal bone disease Hyperphosphatemia and vascular calcification may be additional CVD risks in CKD	See sections on calcium, phosphorus, parathyroid hormone (PTH), and vitamin D.	**Mineral Imbalance*** CKD may affect mineral balance: • **Vitamin D** may be reduced as the kidneys play a key role in its activation. • **Serum calcium** may be low due to reduced vitamin D. • **Parathyroid hormone (PTH)** may be high, as low serum calcium stimulates the parathyroid gland to secrete additional PTH. • **Serum phosphorus** may remain in the normal range as a result of higher PTH levels. As eGFR decreases serum phosphorus may increase. Parathyroid gland • maintains serum calcium • has an indirect role in maintaining serum phosphorus PTH increases • calcium resorption from bone and increases intestinal calcium absorption by stimulating the enzyme (1-alpha-hydroxylase) in the kidney responsible for the final activation of vitamin D • urinary excretion of phosphorus

* Therapeutic use of phosphate binders and Vitamin D analogs described under interventions are for informational purposes and should not be interpreted as evidence-based recommendations. Existing guidelines on treatment of MBD are largely based on observational data.

V. Preventing, Monitoring, and Treating Complications, *(cont.)*

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION*
Mineral and Bone Disorder, *(cont.)*		**Mineral Imbalance, *(cont.)***
		PTH levels may be lowered through • supplementation with vitamin D (vitamin D and PTH may be inversely related) • phosphorus restriction
		Bone Disorders
		It may be difficult to distinguish between different bone disorder types without a bone biopsy. Depending on the type of renal bone disease, calcium, phosphorus, and iPTH may be normal, decreased, or elevated. • **Secondary hyperparathyroidism** is associated with – high bone turnover – decreased calcium –elevated phosphorus –elevated iPTH • **Osteomalacia** results in – low bone turnover – elevated calcium – normal-to-decreased phosphorus – normal-to-decreased iPTH – normal-to-decreased alkaline phosphatase • **Adynamic bone disease** may be characterized by – low bone turnover – normal-to-elevated calcium – normal-to-elevated phosphorus – normal-to-decreased iPTH – normal-to-decreased alkaline phosphatase • **Mixed bone disease**, as the name implies, has features of both low and high bone turnover.

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V. Preventing, Monitoring, and Treating Complications, *(cont.)*

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION*
Calcium Inadequate calcium may stimulate secondary hyperparathyroidism. Excessive calcium may promote vascular calcification in CKD.	Calcium 8.5 – 10.2 mg/dL Maintain within normal range.	• Calcium-based phosphate-binding medications can increase total daily intake and may elevate calcium. • Use formula to correct calcium with hypoalbuminemia: Corrected calcium (mg/dL) = serum calcium (mg/dL) + 0.8 (4.0 – serum albumin (g/dL))
Phosphorus Hyperphosphatemia may be associated with vascular calcification in CKD. Serum phosphorus levels may be "normal" until CKD is advanced due to PTH and other factors.	Phosphorus 2.7 – 4.6 mg/dL Maintain within normal range.	• Phosphorus binders may be prescribed to lower phosphorus levels: – If prescribed, binders should be taken with meals to decrease absorption of phosphorus from food and beverages. – Calcium acetate and calcium carbonate are common calcium-containing phosphate binders. – Calcium citrate is not recommended as a phosphate binder for CKD patients because it may increase aluminum absorption. – Other binders, used more often in renal replacement therapy, are typically composed of resins (sevelamer carbonate) and earth metals (lanthanum carbonate). • Supplementation with active vitamin D compounds may increase risk for hyperphosphatemia. • Dietary phosphorus restriction is generally recommended: – Phosphorus in food additives may be absorbed more efficiently and should be avoided. These may be identified by reading ingredient lists for words with "phos." – Consider referral to a registered dietitian.

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V. Preventing, Monitoring, and Treating Complications, *(cont.)*

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION*
Parathyroid Hormone (PTH) Secondary hyperparathyroidism (elevated PTH) is associated with • the most common cause of bone disease in CKD • increased risk of vascular calcification Normal PTH < 65 pg/mL	Measured as iPTH (intact PTH) PTH varies by level of kidney function and type of bone disease.	• PTH levels in CKD may be lowered through – dietary phosphorus restriction – supplementation with vitamin D or its analogs – supplementation with calcium
Vitamin D Reduced kidney function results in decreased production and conversion of 25(OH) D to 1,25 (OH)2 D. Imbalances of calcium, phosphorus, and PTH may develop.	Vitamin D ≥ 20 ng/mL Measured as 25(OH)D Maintain within normal range	• Ergocalciferol (vitamin D2) or cholecalciferol (vitamin D3) may be used in early CKD to replete vitamin D. • Active vitamin D (calcitriol) or its analogs (doxercalciferol, paricalcitol, or alfacalcidol) may be used in dialysis patients. • Monitor for hypercalcemia and/or hyperphosphatemia when using supplements. Active vitamin D increases calcium and phosphorus absorption.

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V. Preventing, Monitoring, and Treating Complications, *(cont.)*

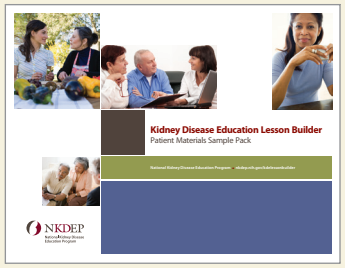
COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION
Hyperkalemia Patients with CKD are at risk for hyperkalemia as a result of • reduced potassium excretion • RAAS antagonist use for blood pressure control • NSAID use for pain control • Intake of high-potassium foods • metabolic acidosis • hyperglycemia	Potassium 3.5 – 5.0 mEq/L Hyperkalemia is usually not seen until CKD is advanced, but may be seen at higher eGFRs in • people with diabetes • people on ACEs/ARBs • people on spironolactone	• Monitor for hyperkalemia with use of RAAS antagonists. • Discontinue use of NSAIDs. • Correction of acidosis may lower potassium. Counsel patients to adhere to sodium bicarbonate therapy, if prescribed. (See Metabolic Acidosis below.) • Manage diabetes to prevent hyperglycemia. **Additional Information** • Caution patients to avoid potassium-containing salt substitutes. Certain low sodium products may have added potassium chloride in place of sodium chloride. • Patients with diabetes and hyperkalemia should treat hypoglycemia with glucose tablets, cranberry juice cocktail or apple juice instead of orange juice or cola.
Metabolic Acidosis Patients with CKD are at risk for metabolic acidosis as a result of reduced • excretion of acid load • bicarbonate synthesis	Normal range: 21 – 28 mEq/L Bicarbonate (CO_2) > 22 mEq/L	• Metabolic acidosis is thought to result in – loss of bone and muscle mass – negative nitrogen balance – increased protein catabolism – decreased protein synthesis • Sodium bicarbonate supplementation may be prescribed to treat acidemia. Monitor blood pressure closely when prescribed, as some patients may experience elevated blood pressure associated with increased sodium load. **Additional Information** Dietary protein, particularly animal protein, is a source of metabolic acid. Serum bicarbonate levels may increase with dietary protein restriction.

V. Preventing, Monitoring, and Treating Complications, *(cont.)*

COMPLICATION AND ITS RELEVANCE	RANGES/GOALS	INTERVENTION
Malnutrition Malnutrition is associated with increased morbidity and mortality in CKD patients. Hypoalbuminemia may result from • inflammation • infection • albuminuria • reduced protein and/or calorie intake • metabolic acidosis or uremia Serum albumin < 4.0 g/dL, at time of initiation of dialysis, is associated with increased morbidity and mortality.	Albumin > 4.0 g/dL Normal range: 3.4 – 5.0 g/dL	• Poor oral health may contribute to inflammation, infection and poor intake. Refer to a nephrologist as needed. May occur even with adequate intake. • Treat acidemia (see Metabolic Acidosis on page 17 or Collaborate with a Nephrologist on page 20). • Refer to a registered dietitian who is familiar with medical nutrition therapy for CKD. **Additional Information** • Malnutrition is common in CKD. Appetite may decrease as eGFR declines. • Appetite may improve in renal failure with adequate renal replacement therapy (i.e., dialysis treatment or kidney transplantation).

VI. Transition to Kidney Failure

ACTION	GOAL	INTERVENTIONS
Prepare Your Patient Early preparation and education about CKD gives the patient time to process the information and prepare both physically and psychologically.	Protect the veins. Discuss options early in the course of CKD. Immunize appropriately.	**Protect blood vessels in the arms for permanent vascular access:** • Avoid venipuncture and intravenous catheter placement proximal to the wrist. • Avoid PICC lines in people likely to progress to ESRD. **It is never too early to discuss the potentially progressive nature of CKD and options for kidney failure:** • Many people have difficulty making choices about treatment modality, vascular access, and initiation of dialysis. • Medicare Part B may cover kidney disease education when the eGFR is less than 30. • Treatment modalities include – kidney transplant – peritoneal dialysis – hemodialysis – management without replacement of kidney function (i.e., no RRT). Some opt for no RRT particularly when the risks and burdens outweigh the potential benefits. Comorbidities and complications are managed medically. **Routinely recommended vaccines should be given if not contraindicated.** Risk-specific recommended vaccines for CKD include pneumococcal and Hepatitis B.

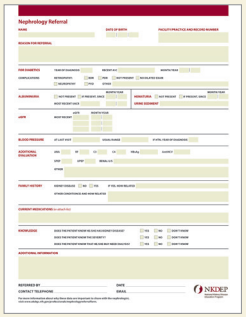


RELEVANT RESOURCE

The KDE Lesson Builder helps providers counsel CKD patients about managing their disease and preparing for renal replacement therapy (RRT). Lessons 4–6 focus on preparing for RRT: nkdep.nih.gov/identify-manage/educate-patients.shtml

VI. Transition to Kidney Failure, (cont.)

ACTION	GOAL	INTERVENTIONS
Collaborate with a Nephrologist Timing of the referral may vary depending on patient status as well as provider experience. Kidney failure is typically defined as an eGFR < 15 mL/min/1.73 m^2 Late referral is associated with • more rapid progression of CKD • worse health status at the time of initiation • higher mortality after starting dialysis • decreased access to transplant	Maintain continuity of care and improve outcomes. Inform the patient that he/she is being referred to a nephrologist and the reason for the referral. Provide basic data with referral which may include • preliminary evaluation (e.g., ultrasound, screening serologies) • patient history including serial measures of eGRF and UACR • other pertinent results	• Patients with CKD and other chronic illnesses benefit from interdisciplinary care. • It may be appropriate to consult with a nephrologist to – assist with a diagnostic challenge (e.g., decision to biopsy) – assist with a therapeutic challenge (e.g., blood pressure, anemia, hyperphosphatemia, secondary hyperparathyroidism, hyperkalemia, metabolic acidosis) – assess rapid decrease of eGFR – treat most primary kidney diseases (e.g., glomerulonephritis) – prepare for renal replacement therapy, especially when eGFR is less than 30 mL/min/1.73 m^2 **Additional Information** • Placement of permanent dialysis access (hemodialysis or peritoneal access) should be planned so that it is functional at the time of initiation. • Patients with advanced CKD may be referred for transplant evaluation prior to starting dialysis.

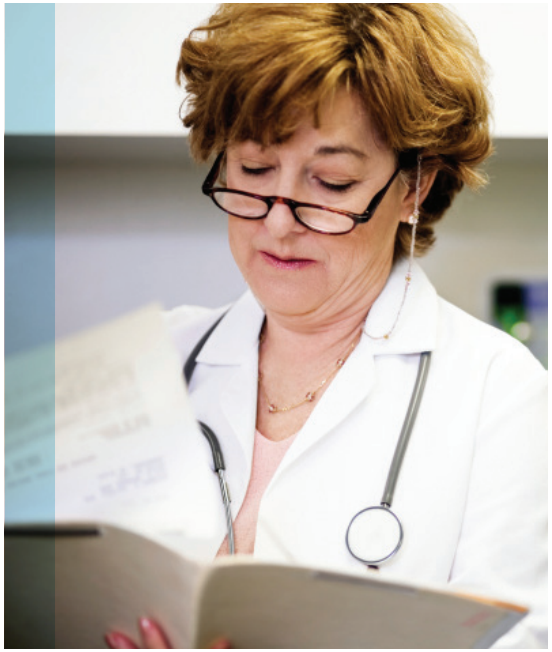


RELEVANT RESOURCE

The Nephrology Referral Form makes it easy to share important patient data with the consulting nephrologist: nkdep.nih.gov/neph-referral

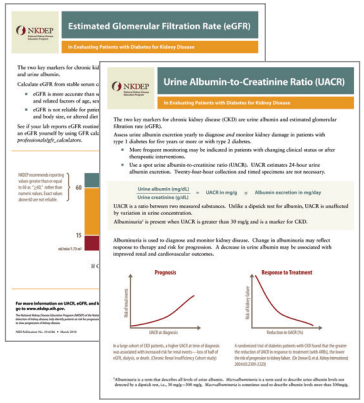
VII. Provider and Patient Resources

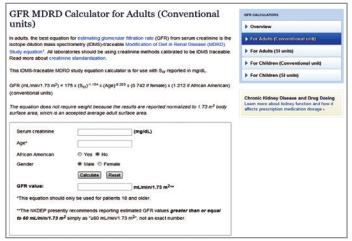
NKDEP offers numerous materials to support providers and patients with CKD. These free materials—designed to provide key information about CKD—are available to download or view from the NKDEP website at nkdep.nih.gov/resources.

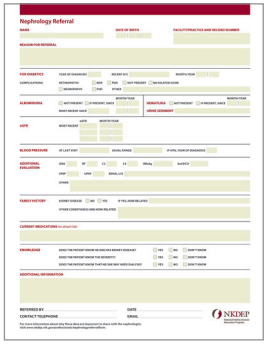


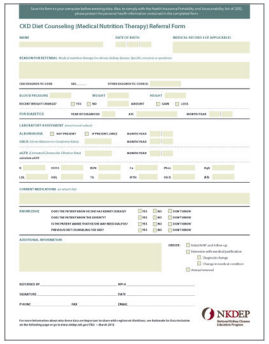
For Providers

- ***Quick Reference on UACR and GFR*** — Provides key information about evaluating patients with diabetes
- ***GFR Calculator for Adults and Children*** **(Online Only)** — For use when estimating kidney function in adults and children. nkdep.nih.gov/gfr-calculators
- ***Nephrology Referral Form*** **(Online Only)** — An interactive PDF with fields for entering key clinical information to help referring physicians share patient information with nephrologists. nkdep.nih.gov/neph-referral
- ***CKD Diet Counseling (Medical Nutrition Therapy) Referral Form*** **(Online Only)** — An interactive PDF with fields for entering key clinical information to help referring physicians share patient information with registered dietitians. nkdep.nih.gov/mnt-referral
- ***Kidney Disease Education Lesson Builder*** **(Online Tool and Sample Pack)** — A tool to help educators create and implement lesson plans for counseling patients with CKD and find patient education resources based on CKD topic. nkdep.nih.gov/kde-lesson-builder





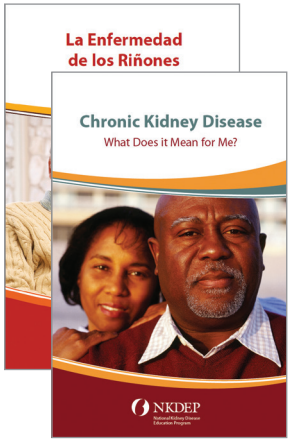


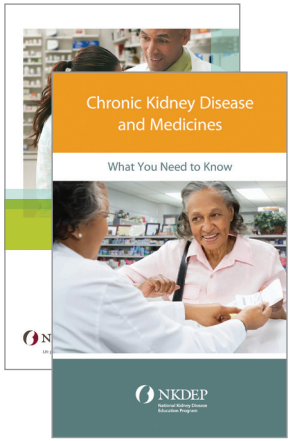


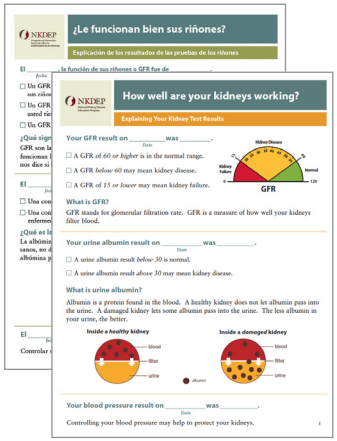
For Patients

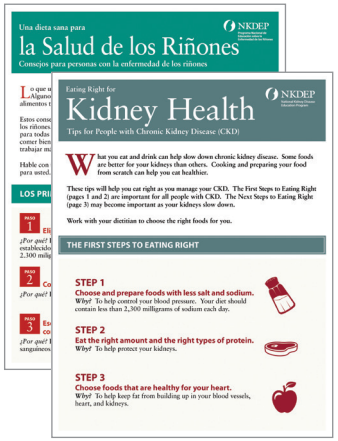
Available in English and Spanish

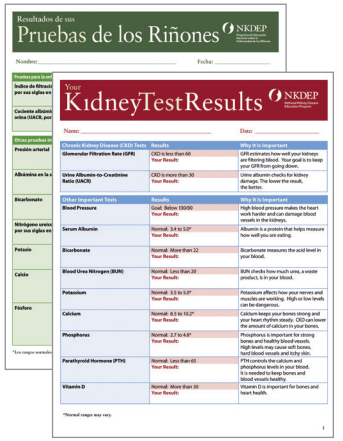
- ***Chronic Kidney Disease: What Does it Mean for Me?*** — Explains the basics of CKD for recently diagnosed patients
- ***Chronic Kidney Disease and Medicines: What You Need to Know*** — Explains how to use medicines to people with CKD
- ***Explaining Your Kidney Test Results: A Tear-off Pad for Clinical Use*** — Easy-to-read sheet to explain GFR and urine albumin
- ***Eating Right for Kidney Health: Tips for People with Chronic Kidney Disease*** — A handout on the basics of nutrition and CKD
- ***Your Kidney Test Results*** — A tool for assessment and education of test results with patients











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