Clinical Guide to Nutrition Care in Kidney Disease

THIRD EDITION

Renal Practice Group
National Kidney Foundation Council on Renal Nutrition

Editors: Janelle E. Gonyea, RDN, LD, FNKF, and Stacey C. Phillips, MS, RDN

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Contents

Frequently Used Terms and Abbreviations

AACE	American Association of Clinical Endocrinologists
AADE	American Association of Diabetes Educators
ACC	American College of Cardiology
ACE	angiotensin-converting enzyme
ADA	American Diabetes Association
ADCES	Association of Diabetes Care & Education Specialists
AF	atrial fibrillation
AGE	advanced glycation end
AHA	American Heart Association
AKI	acute kidney injury
AMDR	acceptable macronutrient distribution ranges
AN	autonomic neuropathy
APD	automated peritoneal dialysis
ARB	angiotensin II receptor blockers
ASPEN	American Society for Parenteral and Enteral Nutrition
AUA	American Urological Association
BG	blood glucose
BIA	bioelectrical impedance analysis
BMD	bone mineral density
BMI	body mass index
BP	blood pressure
BUN	blood urea nitrogen
CAC	coronary artery calcification
CAD	coronary artery disease
CAN	cardiovascular autonomic neuropathy
CAPD	continuous ambulatory peritoneal dialysis
CaSR	calcium-sensing receptors
CCPD	continuous cyclic peritoneal dialysis
CGM	continuous glucose monitoring
CHD	coronary heart disease
CHF	congestive heart failure
CKD	chronic kidney disease
CRP	C-reactive protein
CRRT	continuous renal replacement therapy
CT	computed tomography
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DKD	diabetic kidney disease
DM	diabetes mellitus
DN	diabetic neuropathy
DPI	dietary protein intake
DR	diabetic retinopathy
DRI	Dietary Reference Intake
DXA	dual-energy x-ray absorptiometry
EASD	European Association for the Study of Diabetes
EH	enteric hyperoxaluria
EN	enteral nutrition
ESA	erythropoietin-stimulating agents
ESKD	end-stage kidney disease
FDA	US Food and Drug Administration
FSGS	focal segmental glomerulosclerosis
GA	glycated albumin
GAN	gastrointestinal autonomic neuropathy
GDM	gestational diabetes mellitus
GDP	glucose degradation products
GFR	glomerular filtration rate
GRV	gastric residual volume
GV	glycemic variability
HCFC	high concentrated fructose corn
HDL	high-density lipoprotein
HF	heart failure
HHD	home hemodialysis
IBD	inflammatory bowel disease
IBW	ideal body weight
ICHD	in-center hemodialysis
ICU	intensive care unit
IDWG	intradialytic weight gain
IFG	impaired fasting glucose
IH	idiopathic hypercalcuria
ILE	intravenous lipid emulsion
INR	international normalized ratio
IR	insulin resistance
IRS	insulin resistance syndrome
ISRNM	International Society of Renal Nutrition and Metabolism
KDIGO	Kidney Disease Improving Global Outcomes

KDOQI	Kidney Disease Outcomes Quality Initiative
KRT	kidney replacement therapy
LDL	low-density lipoprotein
LEA	lower-extremity amputation
LVH	left ventricular hypertrophy
MBD	mineral bone disease
MD	maintenance dialysis
MI	myocardial infarction
MNT	medical nutrition therapy
MS	metabolic syndrome
MUAC	mid-upper arm circumference
NFPE	nutrition focused physical exam
NHANES	National Health and Nutrition Examination Survey
NIPD	nocturnal intermittent peritoneal dialysis
NODAT	new-onset diabetes after transplantation
NPH	neutral protamine Hagedorn
OGTT	oral glucose tolerance test
ONS	oral nutrition supplements
PA	physical activity
PAD	peripheral artery disease
PAL	physical activity level
PCR	protein catabolic rate
PD	peritoneal dialysis
PEG	percutaneous endoscopic gastrostomy
PET	peritoneal equilibration test
PH	primary hyperoxaluria
PN	parenteral nutrition
PNA	protein equivalent of nitrogen appearance
PPN	peripheral parenteral nutrition
PRAL	potential renal acid load
PTH	parathyroid hormone
PVD	peripheral vascular disease
RBC	red blood cell
RDN	registered dietitian nutritionist
RFS	refeeding syndrome
RKF	residual kidney function
RR	risk ratio
RTA	renal tubular acidosis
SCCM	Society of Critical Care Medicine
SD	standard deviation
SDI	standard dietary intake
SDS	standard deviation scores
SGA	subjective global assessment
SIADH	syndrome of inappropriate antidiuretic hormone
SMBG	self-monitoring of blood glucose
SO	soybean oil
SPS	sodium polystyrene sulfonate
TLC	therapeutic lifestyle change
TNA	total nitrogen appearance
TPD	tidal peritoneal dialysis
TW	target weight
UL	Tolerable Upper Intake Level
USRDS	United States Renal Data System
UTI	urinary tract infection
VC	vascular calcification
VDR	vitamin D receptor
VLDL	very low-density lipoprotein
WHI	Women's Health Initiative
WHO	World Health Organization
WHR	waist-to-hip ratio

About the Editors

Janelle E. Gonyea, RDN, LD, FNKF, received her dietetics degree from Michigan State University in East Lansing, MI, and completed her dietetic internship at Mayo Clinic Hospital, Saint Marys Campus in Rochester, MN. She has 30 years of experience as a renal dietitian at the Mayo Clinic in Rochester, MN, working with chronic kidney disease patients in all stages of their disease process and across all treatment options. In addition to patient care, she authored medical nutrition therapy (MNT) protocols, chaired committees in charge of developing patient education materials and programming for the Division of Nephrology and Hypertension receiving the Mayo Clinic Department of Medicine Outstanding Education Award and educated countless nephrology colleagues, medical students, nephrology fellows and dietetic interns, holding the rank of Assistant Professor of Nutrition for the Mayo Clinic College of Medicine.

In addition to her clinical dietitian role, Janelle has served on the executive committee of the Council on Renal Nutrition for the National Kidney Foundation as editor of the *RenaLink* professional council newsletter, planning committee chair for the annual Spring Clinical Meeting as well as coordinator for the preconference workshop. Janelle was recognized for her work with the Council on Renal Nutrition by receiving the Susan C. Knapp Excellence in Education Award and Outstanding Service Award. She has also authored numerous articles for patient and professional publications and often speaks at regional and national meetings on various topics related to renal nutrition. Janelle has volunteered as program content creator and reviewer for various patient and professional organizations and received the American Association of Kidney Patients Dietitian Medal of Excellence. Currently she serves as a committee member for the update of the Academy of Nutrition and Dietetics National Kidney Diet, on the Medical Review Committee for the Midwest Kidney Network, as a charter member of the American Kidney Fund's Dietitian Advisory Group and as coeditor for the American Association of Kidney Patients' *AAKP Delicious!* recipe series. Janelle has had a lifelong passion for all things related to nephrology and enjoys the opportunities she has been given to collaborate with others to promote patient advocacy and advancement of care for kidney disease.

Stacey C. Phillips, MS, RDN, has been a registered dietitian nutritionist for over 16 years working for Mercy Health Saint Mary's in Grand Rapids, MI. She received her undergraduate degree from the University of Illinois at Urbana-Champaign, completed her dietetic internship at the Mayo Clinic School of Health Sciences in Rochester, MN, and earned her master's degree in dietetics through Central Michigan University. In her role as a clinical dietitian, Stacey has worked with patients in all stages of chronic kidney disease including kidney transplant recipients and living kidney donors as well as in the areas of general medicine, older adult, oncology, and long-term acute care. Her interest in renal nutrition developed from enjoying this rotation during her own internship due to some dedicated preceptors and through her current work Stacey has mentored over 70 dietetic interns as they rotate through their older adult and renal nutrition rotations.

Outside of her clinical work, Stacey has been involved with the Renal Dietitians Dietetics Practice Group through the Academy of Nutrition and Dietetics in several different roles including the Renal Nutrition Forum Editorial Board, Treasurer, Awards Chair, and Marketing and Communications Chair and was recognized with

the 2021 Renal Dietitians Dietetics Practice Group Outstanding Service Award. She has served as coeditor of the American Association of Kidney Patients' *AAKP Delicious!* recipe series and was awarded the 2019 American Association of Kidney Patients Dietitian Medal of Excellence. In addition, Stacey has been a member of the National Kidney Foundation and served as the Patient Education Editor for the *Journal of Renal Nutrition*. Currently, she enjoys a number of consultant roles including as a subject matter expert for Dietitians on Demand, a Health Coach for the University of Michigan Controlling Hypertension Through Education and Coaching in Kidney Disease (CHECK-D) Study and as a nutrition reviewer for several different professional organizations. She is also a contributor to online medical information, peer-reviewed journals, and Academy of Nutrition and Dietetic publications. With each of these roles, Stacey has enjoyed working with a dynamic group of registered dietitians all with the same mindset of improving the available resources for professionals and patients.

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Preface

Nearly four decades ago, the Renal Dietitians Dietetic Practice Group of the Academy of Nutrition and Dietetics and the Council on Renal Nutrition of the National Kidney Foundation began development of a clinical guide to assist dietetic professionals managing the nutrition care of persons with kidney disease. At that time, renal dietitians were surveyed and they identified three key objectives for the publication. The *Clinical Guide to Nutrition Care in Kidney Disease* should address the following objectives:

1. Contain practical information that is useful in day-to-day clinical practice.
2. Represent a consensus formed by clinical practitioners based on current scientific literature and experience.
3. Focus on characteristics unique to the kidney disease population.

This edition, as well as previous editions, was written with these key objectives in mind.

The *Clinical Guide to Nutrition Care in Kidney Disease* also recognizes that registered dietitian nutritionists (RDNs) who practice in the realm of nephrology nutrition provide both preventive and therapeutic nutrition care for individuals of all ages who present with a variety of kidney disorders, requiring a continuum of treatment options to successfully manage established kidney disease. Consequently, this third edition of *Clinical Guide to Nutrition Care in Kidney Disease* continues to expand content to keep pace with the rapidly evolving field of nephrology. Enduring content areas have been updated to reflect the newly published Kidney Disease Outcomes Quality Initiative (KDOQI) Clinical Practice Guideline for Nutrition in CKD: 2020 Update. Updates also support the efforts of practitioners to achieve goals set forth by the July 2019 Executive Order on Advancing American Kidney Health which focuses on the prevention of progressive chronic kidney disease (CKD) and the promotion of home dialysis modalities as well as kidney transplantation in the event that CKD progresses to the point where kidney replacement therapy is required, ultimately improving medical care and quality of life for individuals with kidney disease.

The number of topics have been expanded to reflect the ever-broadening scope of practice for RDNs employed in outpatient clinics, transplant centers, dialysis facilities, long-term care facilities, and hospitals. New topics found in the third edition include nutritional management of kidney stones, guidelines for patients following plant-based diets to support recommended dietary patterns in the 2020 KDOQI update, CKD and gut health, information regarding herbal and botanical use in patients with transplant as well as malnutrition criteria, nutrition focused physical examination, and an overview of oral nutrition supplements for use in patients with kidney disease to help prevent or treat malnutrition that is commonly identified in this patient population.

This third edition of the *Clinical Guide to Nutrition Care in Kidney Disease* will provide continuing education credits for those who desire to pursue this option and assist RDNs who are preparing to take the credentialing examination to become a Certified Board Specialist in Renal Nutrition. With the aforementioned enhancements, we hope that you will find this third edition of the *Clinical Guide to Nutrition Care in Kidney Disease* to be an invaluable resource as you care for individuals with kidney disease.

A special thank you to previous clinical guide coeditors Karen Wiesen, MS, RDN, LDN, FNKF, Jean Stover, RD, LDN, and Laura Byham-Gray, PhD, RDN, FNKF, for their guidance and support with the third edition.

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SECTION I

Overview of Kidney Disease and Nutrition Assessment

CHAPTER 1

Overview: Pathophysiology of the Kidney

Janelle E. Gonyea, RDN, LD, FNKF, and Brittany Sparks, RDN, CSR

Basic Kidney Anatomy

The kidneys are located symmetrically on either side of the vertebrae, starting at the 12th thoracic vertebra and extending down to the third lumbar vertebra. Each adult kidney measures approximately 11 cm to 12 cm long, 5 cm to 7.5 cm wide, 2.5 cm to 3 cm thick, and weighs approximately 115 g to 170 g. Each kidney has two general regions: the cortex, and the medulla (see Figure 1.1). The medulla is divided into cone-shaped regions called renal pyramids.[1] Blood is supplied to healthy kidneys through the renal artery, which enters the kidney at the hilus. Approximately 20% of cardiac output circulates to the kidneys. This is far more blood flow than necessary to supply needed oxygen to the kidneys, but it is required for excretory function.[2] Urine that has been formed collects in the lower portion of the renal pelvis, which is the expanded upper region of the ureter. It then exits the kidney via the ureter that extends for a length of 22 cm to 30 cm, providing a connection to the bladder.[3]

The basic functional unit of the kidney is the nephron (see Figure 1.2 on page 2). On average, each human kidney contains 900,000 to 1 million nephrons, which are located in both the cortex and the medulla.[4] Within each nephron, there are several well-known microscopic components (see Box 1.1 on page 2).

FIGURE 1.1 | Anatomy of a kidney

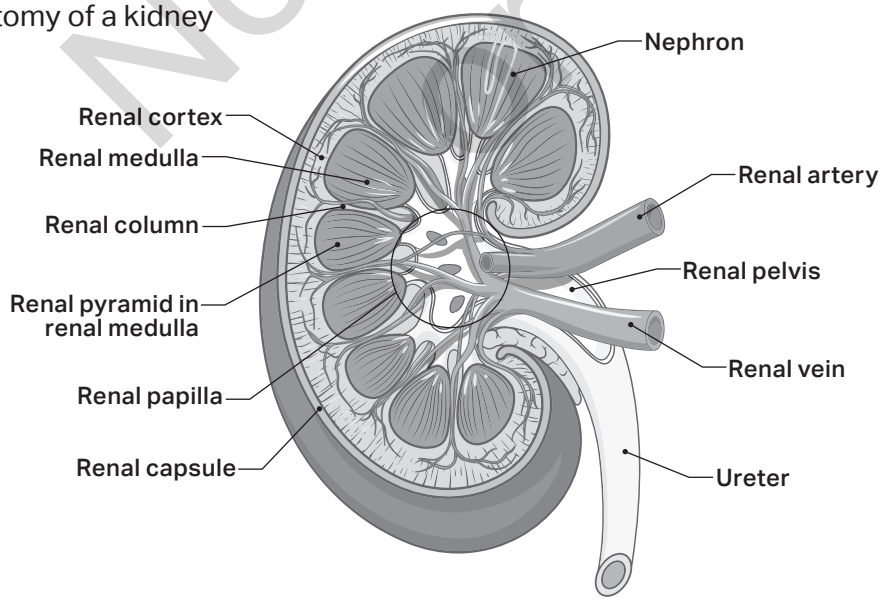
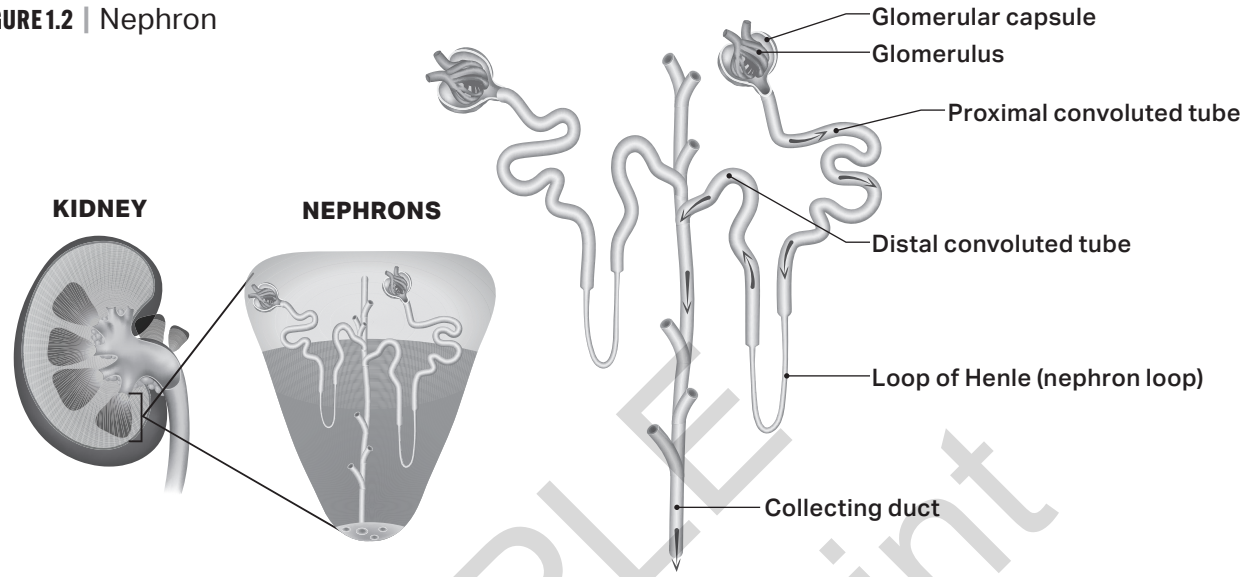


FIGURE 1.2 | Nephron



BOX 1.1 | Functional Units of the Nephron[4]

NEPHRON COMPONENT	FUNCTION
Glomerulus/ Bowman's capsule/ renal corpuscle	Serves as the kidney's filtering unit
	Made of a network of capillaries surrounded by a narrow wall of epithelial cells
	Forms approximately 125 mL/min of filtrate within the glomerulus
	Bowman's capsule, with its basement membrane, encompasses the glomerulus (Together they are known as the renal corpuscle)
Proximal convoluted tubule	Is a direct continuation of the epithelium of the Bowman's capsule
	Controls reabsorption of filtered glucose, amino acids, sodium, bicarbonate, potassium, chloride, calcium, phosphate, water, and other solutes
	Manages secretion of renally excreted drug metabolites
	Produces ammonium
Loop of Henle	Concentrates urine which may be diluted later in the excretory system
	Unable to permeate water—but sodium can be pumped out
	Influences movement of water in or out of the water–permeable collecting duct
Distal tubule	Comprised of three parts: 1. the thick ascending limb of the loop of Henle 2. the macula densa 3. the distal convoluted tubule
	Has ability for active transport of sodium chloride in the thick ascending limb of the loop of Henle
	Concentrates urine by absorbing approximately 99% of the water that is filtered by the kidneys back into the body
	Thick ascending limb of loop of Henle functions are governed by hormones, including vasopressin, parathyroid hormone, and calcitonin
Collecting duct	Contains two types of cells: principal cells and intercalated cells
	Controls reabsorption of sodium chloride and the secretion of potassium via principal cells
	Regulates acid-base balance via secretion of hydrogen and bicarbonate in intercalated cells

Kidney Functions

The major functions of the kidneys can be described as excretory, acid-base balance, endocrine, and fluid and electrolyte balance.

Excretory functions of the kidneys include removing excess fluid and waste products. While approximately 180 L of filtrate pass daily through the kidneys, only 1 to 2 L are removed in the urine each day.[5] The remaining fluid is retained in the body to support tissues. Substances removed with the urine include urea, vitamins and minerals consumed in excess of the body's requirements, and metabolites of some drugs and poisons. If the blood levels of any needed substances are low, the kidneys promote homeostasis by conserving them to maintain levels within narrowly defined limits.

Acid-base balance is maintained through a buffer system, which keeps the blood's pH level at approximately 7.4. Bicarbonate carries hydrogen ions to the kidney, where they are removed from extracellular fluid in the tubules, then reabsorbed in the proximal tubule, and then returned as needed to the bloodstream. In addition, phosphate buffers intracellular fluid, which is concentrated in the tubules as water is removed. Other organic compounds, such as citrate, also support acid-base balance. Finally, metabolites of amino acids may be used to moderate acid or base reactions.[6]

Several hormones are included in the endocrine function of the kidneys. Calcitriol, or 1,25-dihydroxy-vitamin D3, is produced in the kidney and subsequently enhances calcium absorption. In healthy kidneys, the activation of vitamin D and the excretion of excess phosphorus set the stage for maintaining healthy bones (see Chapter 10 for more details).

Anemia of patients with chronic kidney disease (CKD) is related to altered production of erythropoietin (EPO) in the diseased kidney. Normally, EPO acts on bone marrow to increase the production of red blood cells, thus enhancing the transportation of oxygen throughout the body's tissues. In patients with CKD, decreased production of EPO can lead to anemia (see Chapter 2 for more details).

The kidneys play an important role in maintaining fluid and electrolyte balance. Antidiuretic hormone (ADH), also known as arginine vasopressin, is released from the posterior pituitary gland and helps regulate water that is reabsorbed into the body. When fluid volume (blood or other fluids) is low, ADH is secreted and reduces urine flow, increasing the reabsorption of water in the collecting duct. This mechanism maintains the body's osmolality within a very tight range.

When extracellular volume decreases, the perfusion of all body tissues—including the kidney—is reduced. Consequently, the glomerular filtration rate (GFR) is lower, resulting in decreased removal of sodium chloride. This activates the trio of hormones of the renin-angiotensin-aldosterone system to increase blood pressure to maintain adequate tissue perfusion.[7]

A person with advanced kidney function impairment may experience edema, uremia (accumulation of waste products in the blood), metabolic acidosis, hypertension, anemia, bone disease, and increased sensitivity to certain medications, vitamins, minerals, and dietary supplements, which can reach toxic levels in the body.

Types of Kidney Failure

The onset of kidney failure can be sudden, as in acute kidney injury (AKI), or progressive, leading to CKD. Patients with AKI will potentially regain their kidney function with medical treatment of the underlying cause of insult. However, if a decrease in GFR or an increase in serum creatinine persists for more than 3 months after the diagnosis of AKI, the kidneys are not expected to recover, resulting in CKD. Typically, CKD cannot be reversed but may be stabilized with certain etiologies. Additional challenges for patients with significant proteinuria should also be addressed. Medical nutrition therapy (MNT) is important in both the management of AKI and CKD and will be discussed in detail in Chapters 3 and 4.

ACUTE KIDNEY INJURY

AKI generally has a rapid onset of symptoms occurring over a period of hours to days. AKI may start with a small increase in serum creatinine and progress to reduced urine volume (oliguria) and ultimately to a complete absence of urine output (anuria).[8] AKI can occur in previously healthy kidneys. With proper nutrition management and appropriate dialytic support, ranging from intermittent hemodialysis to continuous venovenous hemodiafiltration, the kidneys can often repair themselves (see Chapter 4).[9] However, AKI can often be superimposed on underlying CKD secondary to diabetes, hypertension, and other comorbidities. In this scenario, the goal is to return kidney function to baseline by removing the offending agent and avoiding chronic dialysis. AKI is associated with a high degree (40%–70%) of morbidity and mortality, despite advancing technology in kidney replacement therapy (KRT).[10,11] AKI is also associated with increased risk for development of CKD, even when there is complete recovery of kidney function.[10-13]

In the United States, AKI most commonly occurs in hospitalized and critically ill patients and is often secondary to ischemic events and consequent injury to the kidney. However, AKI can arise from many different etiologies. AKI can be characterized by the type of underlying insult to the kidney and is categorized as either prerenal, intrinsic or tubulointerstitial, or postrenal.[14-16] Prerenal AKI results from decreased kidney perfusion in an otherwise normal kidney, perhaps due to altered cardiac function and altered glomerular blood flow; with treatment, it can be quickly reversed. AKI may have a kidney origin due to underlying structural or functional changes in the kidney, and postrenal etiologies of AKI may include urethral strictures, tumors, clots, or stones restricting flow to or from the urinary tract.[14,16] Identifying where the damage may be occurring within the kidney is key to treatment (see Chapter 4 for a more detailed discussion on AKI).

CHRONIC KIDNEY DISEASE

In 1997, the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF-KDOQI) began publishing evidence-based clinical practice guidelines. These guidelines were developed and implemented by physicians and health care providers within the nephrology community. In 2002, NKF-KDOQI outlined a standard for classifying stages of CKD.[17] A decade later, the Kidney Disease: Improving Global Outcomes (KDIGO) organization offered an update to the initial guidelines. The classification for CKD appears in Table 1.1.[18]

TABLE 1.1 | Stages of Chronic Kidney Disease[18]

Stage	Glomerular filtration rate (mL/min/1.73 m²)	Description
G1	≥90	Kidney damage with normal or increased glomerular filtration rate (GFR)
G2	60-89	Mild decrease in GFR
G3a	45-59	Mild-to-moderate decrease in GFR
G3b	30-44	Moderate-to-severe decrease in GFR
G4	15-29	Severe decrease in GFR
G5	<15	Kidney failure

It is important to note that some decrease in kidney function is a normal part of aging. CKD stage 1 and even CKD stage 2 may be observed in otherwise healthy individuals aged 60 years and older. However, even in older adults, reduced kidney function should be monitored to allow appropriate intervention if the decrease in GFR continues into later stages of CKD.[19]

Patients with CKD continually lose GFR over a period of months to years. In many of the underlying conditions, progression of the disease continues even if the kidney is no longer exposed to the initial insult. Eventually, CKD may progress to stage 5, requiring KRT, such as dialysis or transplantation, to sustain life (see Chapters 5, 6, 7 and 8 for more details).

Nephrotic Syndrome

Nephrotic syndrome (NS) is one of the most serious challenges in clinical nephrology.[20] NS is not a disease but rather a collection of symptoms that may be recognized in patients with CKD of several different etiologies. Box 1.2 shows the clinical characteristics of NS.[21] Nephrotic range proteinuria is a common characteristic of NS, which is classified as greater than 3 g urinary protein loss in a 24-hour time frame. With NS, fundamental alterations of the kidney's glomerular basement membrane allow persistent loss of large amounts of protein and other large molecules to pass into the urine. Normal urinary protein losses are approximately 150 mg/d.[22] Conditions often associated with NS include diabetes mellitus, glomerular diseases, amyloidosis, minimal change disease, and focal segmental glomerulosclerosis (FSGS).[22]

BOX 1.2 | Clinical Characteristics of Nephrotic Syndrome

- Proteinuria (>3 g/d of urinary protein losses, with proportionally lesser amounts for children)
- Hypoalbuminemia
- Hypertension
- Hyperlipidemia
- Edema

A urinalysis is typically performed to determine the type and the amount of protein in the urine. This test helps to specify the kind of kidney damage that may be occurring and the necessary treatment. Low-molecular-weight protein losses are often associated with etiologies that affect the kidney tubules and often do not reach nephrotic range proteinuria.[22]

"Albuminuria" is the term used to describe when albumin, a large-molecular-weight protein, is lost in the urine at levels higher than 30 mg/d. Albuminuria is classified as either moderately increased albuminuria with urine albumin levels between 30 mg/d and 300 g/d or severely increased albuminuria, when levels are more than 300 mg/d. These terms are now being used in place of microalbuminuria and macroalbuminuria.[22] Albuminuria is considered to be problematic as it is associated with increased incidence of all-cause mortality, left ventricular hypertrophy, stroke, and vascular calcification.[22] Albuminuria typically results from damage to the filtration barrier and can often be seen in patients with diabetes mellitus and NS.[16]

Since proteinuria, including albuminuria, represents such a high risk factor for cardiovascular disease and CKD progression, the treatment of NS must include reduction of urinary protein losses. Medical intervention may involve corticosteroids and immunosuppressive agents in several specific etiologies. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may also be used to reduce urinary protein losses and control blood pressure and fluid balance. The latter agents may be prescribed to reduce proteinuria even when blood pressure is normal.[21]

The etiology of hyperlipidemia in NS is related to excess production and reduced catabolism of apolipoprotein B–containing lipoproteins (including chylomicrons and very low-density lipoproteins). Some patients present with elevated levels of cholesterol and triglycerides.[23] Hyperlipidemia is associated with an increased risk for vascular disease and, therefore, hydroxymethylglutaryl coenzyme A reductase inhibitors are often used for lipid control. With some disorders, particularly FSGS, there may be a proteinuric factor, and the removal of this factor may help manage proteinuria and subsequent hyperlipidemia.[24]

MNT for NS includes avoiding excessive protein intake, as large protein loads can exacerbate proteinuria. For those with CKD stage 1 and stage 2 with NS, a protein intake of 0.8 g/kg/d of body weight is recommended.[25,26] Refer to Chapter 3 for more information on recommended protein needs for treating patients with CKD stages 3 through 5. Sodium restriction can be beneficial. One study recommends limiting sodium to 100 mmol/d or less than 2,300 mg/d in those with proteinuria; however, the overall goals should be

based on fluid status and the need to control edema.[27] Potassium, other electrolytes, and minerals need to be monitored, and the patient's diet should be individualized. Monitoring for vitamin D insufficiency and deficiency in patients with NS is important, as studies have shown increased risk due to increased loss of vitamin D–binding proteins in the urine.[27-30] In patients with nephrotic range proteinuria, vitamin D supplementation with either cholecalciferol, ergocalciferol, or other safe and effective 25-hydroxyvitamin D precursors may be necessary to maintain adequate serum levels.[27] Nutrition therapy for the associated hyperlipidemia may not normalize serum cholesterol levels, and pharmacological therapy, as previously described, is generally required.[25,26,30,31] More research and clinical trials are needed to identify optimal treatment for NS, especially for vitamin and mineral supplementation.

Summary

This chapter has provided an overview of normal kidney function and various consequences of kidney disease, along with many potential etiologies for compromise in kidney function including AKI, CKD, and NS. More detailed information regarding medical treatment and MNT required at these various stages will be found throughout this publication.

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