Improving Outcomes for Patients with Chronic Kidney Disease: Part 2

Addressing disease complications and treatment for kidney failure: a clinical review.

ABSTRACT: Coping with chronic kidney disease (CKD) is challenging for many people, since symptoms often don't appear until the disease is advanced and the patient is close to requiring dialysis. This two-part article aims to provide nurses with the basic information necessary to assess and manage patients with CKD. Part 1, which appeared last month, offered an overview of the disease, described identification and etiology, and discussed ways to slow disease progression. Part 2 addresses disease complications and treatment for kidney failure.

Keywords: chronic kidney disease, collaborative care, end-stage kidney failure, end-stage renal failure, interdisciplinary care, kidney disease

In Part 1 of this article, which appeared last month, we offered an overview of chronic kidney disease (CKD), described its identification and etiology, and discussed ways to slow disease progression. Here, in part 2, we address disease complications and treatment for kidney failure. As in part 1, the case study of Anna Lowry, a 49-year-old woman with CKD, will be used for illustration, offering nurses specific guidance in helping patients to better understand and manage their CKD. (This case is a composite based on the authors' experience.)

COMPLICATIONS OF CKD

As kidney function declines, fewer functioning nephrons remain. The complications associated with CKD are complex, and may include anemia, hyperkalemia, hypoalbuminemia, metabolic acidosis, and abnormal mineral metabolism and bone disease. Laboratory work may show multiple metabolic abnormalities. Yet most people don't feel any different until their CKD is quite advanced. As noted in part 1, dietary choices can affect many of the metabolic abnormalities associated with CKD. Thus referral to a registered dietitian who is knowledgeable about CKD may help in managing complications.

There are many symptoms, signs, and laboratory values that must be tracked in patients with progressive CKD. The unique nature of the nurse–patient relationship may allow nurses to pick up on symptoms and signs that the primary care provider might have missed—particularly nonverbal behaviors and cues. It's essential for nurses to alert primary care providers

to any subtle changes in a patient's condition, psychosocial issues, patient concerns, abnormal laboratory test results, or trends in laboratory values. (See *Case Study: Metabolic Complications.*)

Anemia. In the general population, a hemoglobin level of less than 13 g/dL in men and less than 12 g/dL in women indicates anemia.[1,2] But the optimal target hemoglobin level for people with CKD is currently unknown.

Anemia may occur early in CKD and is generally due to inadequate synthesis of the hormone erythropoietin by the damaged kidneys. The prevalence of anemia increases as the glomerular filtration rate (GFR) declines, affecting nearly 50% of patients with an estimated GFR (eGFR) of less than 30 mL/min/1.73 m^2.[3] Among patients with advanced CKD, there is evidence that the prevalence of anemia is higher in those with diabetes than in those without diabetes.[4]

CKD-associated anemia is generally normochromic and normocytic.[5] That said, identifying and correcting other causes of anemia (such as iron deficiency) is necessary.[5] Assessing iron status in CKD requires a complete blood count and checking iron indices, including serum iron level and total iron-binding capacity (see Table 1[6-9]). These two results are used to determine the transferrin saturation percentage, which reflects available iron. The serum ferritin level is used to assess stored iron. Optimal target levels of serum iron, total iron-binding capacity, and serum ferritin for people with CKD are unknown. Furthermore, they are affected by inflammation,[10] which is common in CKD,[11] making results more difficult to interpret. The absolute reticulocyte count may also be used to differentiate the cause of anemia or to monitor response to treatment.[8]

Ruling out other causes of anemia, including vitamin-deficiency anemia, may also be important. Although megaloblastic anemia is not commonly seen with CKD, some people with diabetes who have taken metformin for years may be vitamin B_{12} deficient. Metformin reportedly decreases absorption of this vitamin.[12] Both B_{12} and folate levels may be lower than normal with metformin use.[13] Metformin is contraindicated in patients with an eGFR below 30 mL/min/1.73 m^2.[14]

Many other factors may contribute to inadequate iron stores in people with CKD. As their GFR declines, patients may lose interest in high-protein foods. Hepcidin, a hormone that plays a key role in controlling iron levels, regulating iron absorption from the gut, and mobilizing stored iron, accumulates in CKD,[15] and this may result in reduced serum iron levels. Inflammation may also play a significant role in reducing iron absorption.[5]

Painting by Vivian Jay, MSN, FNP-BC, www.ebbandflowwatercolors.com.

Since iron deficiency is common in CKD, iron status and hemoglobin levels should be checked before the addition of any iron supplements to the regimen.[5] Supplemental iron is available in both oral and IV formulations. Absorption of oral iron supplements may be reduced by the intake of caffeinated beverages, supplemental calcium or calcium-containing antacids, and H_2-receptor blockers or proton pump inhibitors.[16] Iron supplements may cause unwanted gastrointestinal effects such as heartburn or nausea. Beginning with half the recommended dosage and gradually increasing to the full dosage may help.[16] Patients may also have fewer adverse effects with a different preparation, or by taking iron with food, in divided doses, or along with stool softeners.[16] Injectable erythropoiesis-stimulating agents are used infrequently in treating CKD.

Hyperkalemia. Potassium excretion is regulated by the renin–angiotensin–aldosterone system (RAAS). Perturbations of this system may result in hyperkalemia. Potassium levels tend to increase as GFR declines.[17] Nearly half of patients with an eGFR less than 30 mL/min/1.73 m^2 have serum potassium levels of 4.5 mEq/L or greater.[3] RAAS antagonists may increase the risk of hyperkalemia. Potassium-sparing medications, dietary intake, and transcellular shifts may also affect serum potassium levels.

Several factors can cause potassium to shift between the intracellular and extracellular compartments. Insulin tends to move potassium into the cells; therefore, insulin deficiency can result in hyperkalemia.[18] Metabolic acidosis, characterized by an excess of hydrogen ions in the plasma, may drive potassium out of the cells, as hydrogen ions are buffered intracellularly.[18] As a result, treating both hyperglycemia and acidemia may lower serum potassium.[18] In some patients with CKD, treating acidosis may allow the continued use of RAAS antagonists.[19]

Patients with hyperkalemia should be counseled to limit foods that are higher in dietary potassium and to read ingredient lists in order to avoid foods that contain potassium chloride. Beginning in July 2018, food manufacturers will be required to include potassium on the Nutrition Facts label.[20]

Hypoalbuminemia occurs in CKD as a result of multiple factors. Both acute and chronic inflammation are associated with reduced albumin synthesis.[21] Loss of albumin in the urine in large quantities is associated with reduced serum albumin levels. Metabolic acidosis,[22] insulin resistance,[23] and a decrease in intake of high-protein foods[24] may also contribute to low serum albumin. One large study of patients on maintenance hemodialysis found that a serum albumin level of greater than 3.8 g/dL was associated with reduced mortality risk, with the lowest such risk seen at levels of 4.4 g/dL or greater.[25] Unfortunately, another study found that only 11% of new dialysis patients had serum albumin levels of 4 g/dL or greater.[26]

Metabolic acidosis is usually defined as a serum bicarbonate level of less than 22 mEq/L. The prevalence of decreased serum bicarbonate increases as

Case Study: Metabolic Complications

Anna Lowry, age 49 at initial visit, has type 2 diabetes and a history of gestational diabetes at age 30.

Current status:
- Her eGFR is below 60 mL/min/1.73 m^2.
- She is taking an ARB.
- Her potassium level is slightly above normal.

Medications:
- Insulin NPH/REG 70/30, 45 units twice daily
- **Losartan 100 mg daily**
- Simvastatin 40 mg daily
- Furosemide 20 mg daily
- Baby aspirin

Test	Initial	6-Month Follow-Up	12-Month Follow-Up	18-Month Follow-Up	Reference Range* or Target
HbA_{1c},%	10.1	9.4	8.7	9.2	< 8
Blood pressure, mmHg	138/67	150/92	136/77	145/83	< 140/90
Creatinine, mg/dL	1.1	1.3	1.6	1.7	0.8–1.3
eGFR, mL/min/1.73 m^2	**53**	**44**	**34**	**32**	**> 60**
UACR, mg/g	2,823	3,107	3,542	3, 312	< 30
Potassium, mEq/L	--	**4.2**	**5.1**	**5.2**	**3.5–5**
HC03, mEq/L	--	--	**19**	**20**	**21–32**
Phosphorus, mg/dL	--	--	--	**4.5**	2.7–4.6
Calcium, mg/dL	--	--	--	**8.7**	8.5–10.2

* Reference ranges may vary.

Ms. Lowry's estimated glomerular filtration rate (eGFR) has continued to decline and is now 32 mL/min/1.73 m^2. As a result, she's beginning to show signs of metabolic decompensation. Her serum potassium level is slightly elevated at 5.2 mEq/L and her serum bicarbonate level is slightly lower than normal at 20 mEq/L. Her serum phosphorus and calcium levels are within the normal ranges.

The nurse alerts the primary care provider to Ms. Lowry's borderline potassium and bicarbonate levels and suggests these be retested at the patient's next visit. The primary care provider decreases the dosage of losartan to 50 mg daily, adds sodium bicarbonate 650 mg twice a day, increases furosemide to 40 mg daily, and orders retesting of potassium and bicarbonate levels at the next visit. The nurse explains to Ms. Lowry that her medications have been adjusted in order to address her abnormal potassium and bicarbonate levels.

Table 1. Assessing Iron Status[6-9]

Laboratory Parameter	Reference Range[a]	Notes
Serum iron level	60–170 mcg/dL	Level may decrease with iron deficiency and inflammation.
Total iron–binding capacity (TIBC)	240–450 mcg/dL	• Level may increase with iron deficiency. • Level may decrease with inflammation.
Transferrin saturation (TSAT) measures iron available for erythropoiesis.	• Normal: 20%–50% • Early functional iron deficiency: < 16%	TSAT = serum iron/TIBC × 100
Serum ferritin level measures the amount of stored iron.	Normal: • Men, 18–270 ng/mL • Women, 18–160 ng/mL Depleted iron stores in patients with chronic kidney disease (both sexes): • < 100 ng/mL in non–dialysis-dependent and peritoneal dialysis patients • < 200 ng/mL in hemodialysis patients	Level may increase with inflammation or infection.

[a] Reference ranges are for the general population unless otherwise noted.

GFR declines.[27] Nearly a quarter of patients with an eGFR below 30 mL/min/1.73 m^2 have a serum bicarbonate level of 20.5 mEq/L or less.[3] Chronic metabolic acidosis is associated with accelerated muscle degradation, reduced albumin synthesis, exacerbation of metabolic bone disease, impaired glucose tolerance, increased inflammation, and accelerated CKD progression.[28] Animal protein is a source of acid load, while fresh fruits and vegetables are not. Serum bicarbonate levels may increase as dietary protein intake decreases.[29]

Interventions to treat chronic metabolic acidosis include an adequate (but not excessive) intake of animal protein and a supplemental base, such as sodium bicarbonate.[28] It's important to note that one 650-mg tablet of sodium bicarbonate has 178 mg of sodium. Reemphasize dietary salt restriction when sodium bicarbonate is prescribed. Some patients may need an added diuretic to help remove the extra sodium.[28]

Abnormal mineral metabolism and bone disease. Some CKD patients may have low levels of 25-hydroxyvitamin D,[30] which can trigger complications that affect bone strength and increase the risk of vascular calcification.[31, 32] Serum calcium levels also decrease as a result of low vitamin D levels.[33] Serum phosphorus levels may be within the normal range until CKD is advanced.[33]

As eGFR declines, the prevalences of hypocalcemia and hyperphosphatemia increase.[3, 33] Over 19% of patients with an eGFR below 30 mL/min/1.73 m^2 have a serum calcium level of 8.9 mg/dL or less, and nearly 30% have a serum phosphorus level of 4.7 mg/dL or more.[3] The systemic disorders of mineral and bone metabolism associated with CKD are reflected in abnormalities in calcium, phosphorus, parathyroid hormone, and vitamin D metabolism. These derangements result in abnormalities in bone turnover, mineralization, volume, and linear growth or strength.[33] They may be associated with vascular or other soft tissue calcification.[34]

Bone disease in people with CKD is complex, and interpreting laboratory test results is difficult. Although observational data support the correction of the metabolic abnormalities, there is limited high-quality evidence to support intervention. Phosphorus restriction may be implemented while trying to maintain adequate protein intake.[30] Phosphorus-binding medications are generally prescribed with meals in order to prevent phosphorus absorption. Low 25-hydroxyvitamin D levels may be treated with ergocalciferol or cholecalciferol, although further trials are needed to confirm the benefits.[31] Elevated parathyroid hormone levels may be managed with vitamin D and phosphorus restriction.[31] Most adults exceed the recommended dietary allowance of 700 mg/day for phosphorus.[35] According to 2011–2012 National Health and Nutrition Examination Survey (NHANES) data, the average phosphorus intake was 1,393 mg/day.[36]

Depending on the food source, phosphorus absorption varies. Between 10% and 60% of phosphorus found naturally in protein-rich foods (such as meat, poultry, dairy, nuts, seeds, dried beans, and whole grains) is absorbed.[37] Between 80% and 100% of inorganic phosphorus, which is added to many

packaged and processed foods, is absorbed.[37] For example, colas contain phosphoric acid. Sodium phosphates are often added to poultry and pork products to enhance flavor or preserve tenderness. Cereals fortified with calcium may contain calcium phosphate.

Patients should be counseled to check ingredient lists for words containing "phos"—such as phosphorus, sodium phosphate, or pyrophosphate—and to avoid foods that contain such ingredients. Because protein-rich foods tend to contain significant amounts of phosphorus, reducing consumption of such foods may also help in reducing phosphorus intake.

PREPARING FOR KIDNEY FAILURE

Coping with CKD and kidney failure is challenging for many people, since symptoms don't often appear until the disease is advanced and the patient is close to requiring dialysis. Many patients exert great effort

Case Study: Preparing for Kidney Failure

Current status, two years after diagnosis:

- Ms. Lowry is upset because a month ago she learned she has progressive kidney disease.
- She reports frequent low blood glucose levels.
- She has stopped following any dietary restrictions and her HbA_{1C} is lower.
- She is still taking her medications.

Test	Initial	Two Years Later	Reference Range* or Target
HbA_{1c},%	10.1	6.9	< 8
Blood pressure, mmHg	138/67	160/90	< 140/90
Creatinine, mg/dL	1.1	2.6	0.8–1.3
eGFR, mL/min/1.73 m^2	53	26	> 60
UACR, mg/g	2,823	5,444	< 30
Hemoglobin, g/dL	12.1	11.6	12–16
Potassium, mEq/L	3.7	4.6	3.5–5
Albumin, g/dL	2.6	2.2	3.4–5
25-hydroxyvitamin D, ng/mL	12	28	20
Calcium, mg/dL	8.7	8.3	8.5–10.2
Phosphorus, mg/dL	3.7	4.9	2.7–4.6
iPTH, pg/mL	--	137	< 65

* Reference ranges may vary.

Over the course of two years, Ms. Lowry's estimated glomerular filtration rate has dropped from 53 to 26 mL/min/1.73 m^2. Upon learning that her disease has progressed despite her best efforts to adhere to the treatment regimen, she becomes very upset. Frustrated, she stops following the recommended dietary restrictions. Despite this, her glycated hemoglobin level continues to decrease.

The nurse listens patiently as Ms. Lowry expresses her turmoil, despair, fear, and sadness about the progression of her chronic kidney disease. The nurse acknowledges these emotions, expresses empathy, and explains that Ms. Lowry's feelings are normal. The nurse encourages her to learn more about kidney failure and its treatment so that she can regain some sense of control.

Knowing that Ms. Lowry has been reluctant to tell her husband and son about her illness, the nurse asks Ms. Lowry to invite them to the next office visit, so they can all discuss her care together. She reminds Ms. Lowry that her family will want to be there to support her and help her make difficult treatment decisions. The nurse also

- educates Ms. Lowry on kidney failure and its treatment options, helping her to understand how each treatment might affect her life.
- refers her to a booklet that explains kidney failure treatment options. (For a copy, visit www.niddk.nih.gov and search using the phrase "Kidney Failure: Choosing a Treatment That's Right for You.")
- helps Ms. Lowry find a local kidney disease education class where she can learn more about treatment options and hear from patients who have been through the decision-making process.
- helps her schedule a visit to a nearby dialysis center so she can see firsthand what it's like.
- asks about her immunization status, emphasizing the importance of keeping vaccinations up to date.

in adhering to all treatment recommendations, yet still experience CKD progression. Furthermore, both the disease itself and treatment with dialysis are complicated matters. Patients may have difficulty understanding the implications of changes in their health status. It's not uncommon for patients to experience grief, fear, or depression.

Historically, the health care community has not done a good job in educating CKD patients. An analysis of 1994–1998 and 1999–2000 NHANES data showed that fewer than 20% of patients with both moderately decreased kidney function (an eGFR of 30 to 59 mL/min/1.73 m^2) and albuminuria (a urine albumin-to-creatinine ratio greater than 30 mg/g) had ever been told by a physician that they had "weak or failing kidneys."[38] Such delayed awareness leaves many patients with little time to prepare for kidney failure, leaving them limited options when they face decisions about treatment. As one reflection of inadequate preparation, consider that in 2013 more than 80% of people started hemodialysis with a temporary vascular access (catheter).[39]

Many nurses aren't comfortable discussing CKD or dialysis with patients,[40,41] and may defer such discussions to the nephrologist. This is a missed opportunity, because it's both appropriate and helpful for nurses to initiate these conversations. A new provider may not know the patient well, whereas the nurse in the diabetes or hypertension clinic who has been involved in managing the patient's conditions will have established a trust that can make education more effective. Nurses can help patients to better understand CKD and begin accepting and coping with changes in their health status. (See *Case Study: Preparing for Kidney Failure*.)

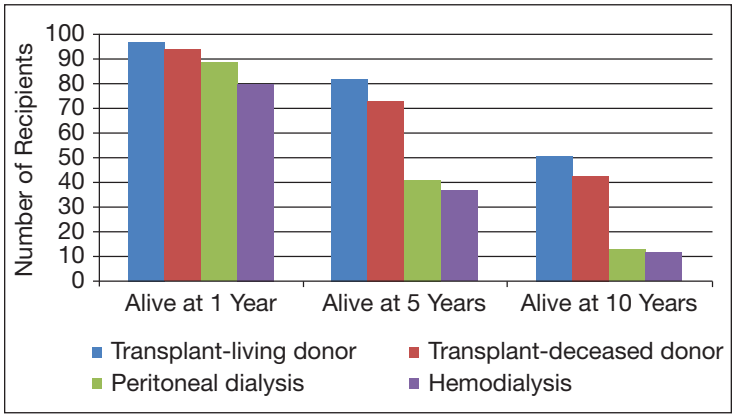
TREATMENT CHOICES FOR KIDNEY FAILURE

There are four options for treating kidney failure. Three involve kidney replacement therapy, including kidney transplantation, peritoneal dialysis, or hemodialysis (either in a dialysis center or at home). The fourth option involves supportive care without transplantation or dialysis.

Transplantation. In general, a kidney transplant is associated with the best quality of life and survival (see Figure 1[42]). To receive a kidney transplant, a patient must be healthy enough to endure a surgery that can last several hours; have access to a donated kidney, either through a living donor or by being on the waiting list for a deceased donor kidney; and be willing to take antirejection medications daily and to have routine follow-up appointments for the rest of her or his life. Although antirejection medications suppress the immune system, organ rejection remains a possibility. With a functioning transplant, dialysis is not needed, and a near-normal diet can be followed.

Kidney transplantation is a treatment, not a cure. The transplanted kidney will likely work very well for a time—according to recent data trends analyses, 92% of deceased donor and 97% of living donor kidneys continue to work at one year following transplantation, and 47% of deceased donor and 62% of living donor kidneys are still working 10 years later[39]—but eventually it is likely to fail. The outcome improves if the donor and the recipient are ABO blood-type compatible and a match for human leukocyte antigens. Pretransplantation evaluation can take several months and typically includes a comprehensive assessment to check for the presence of any conditions that might place the patient or the transplanted kidney at risk (such as severe coronary artery disease or cancer). Eligibility criteria vary from facility to facility, with some more willing to include patients with a higher body mass index.

Figure 1. Survival Rates at One, Five, and 10 Years for Patients Opting for Kidney Transplantation or Dialysis



Survival rates for recipients of a living donor kidney are higher at one, five, and 10 years following the transplant compared with those for recipients of a deceased donor kidney, peritoneal dialysis, or hemodialysis. Survival rates for recipients of a kidney from either type of donor are higher at one, five, and 10 years following the transplant compared with those for recipients of either peritoneal dialysis or hemodialysis. Disparities in survival rates increase over time. Adapted with permission from Johns Hopkins University, PREPARED (Providing Resources to Enhance Patients' Readiness to Make Decisions About Kidney Disease) Study Research Team. *Preparing for Kidney Treatment: You Have a Choice*. 2011.[42]

Peritoneal dialysis may be a choice for a patient who has no contraindicating abdominal pathology (such as extensive abdominal surgery), wants to do in-home treatment, is willing to perform the treatment daily, and has room to store the necessary supplies. Patients using peritoneal dialysis usually don't require vascular access, but do require minor surgery for abdominal catheter placement.

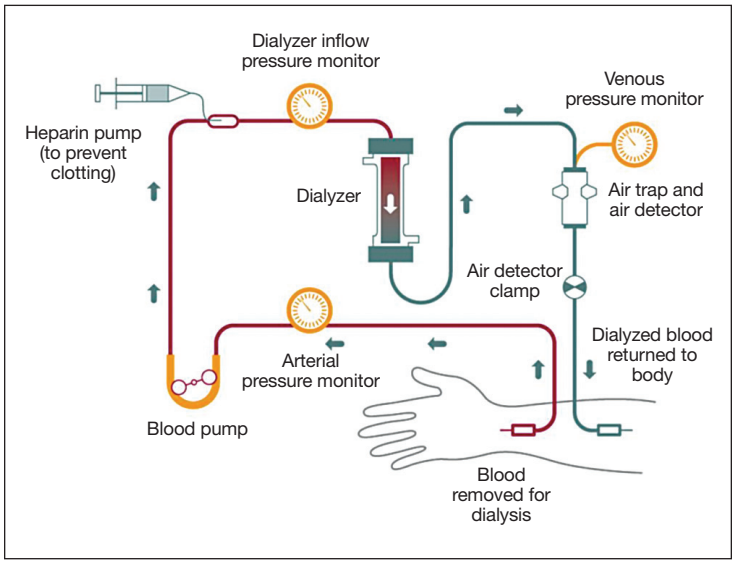
In peritoneal dialysis, the peritoneal membrane is used as a semipermeable filter, replacing the kidneys. In a peritoneal dialysis exchange, a dialysis solution (the dialysate) flows through the catheter into the abdominal cavity, where it remains for a prescribed

period of time known as the dwell time. Through the process of diffusion, waste products move down the concentration gradient from the blood in the peritoneal capillaries into the dialysate. The efficiency of this clearance is determined by the concentration gradient; the size of the solute; and the permeability of the peritoneal membrane, which can vary over time. The dialysate includes an osmotic agent that draws fluid into the peritoneal cavity, removing water and producing some additional clearance by bulk flow. At the end of the dwell time, the solution is drained out through the catheter. The continuous nature of peritoneal dialysis allows the patient to reach equilibrium, avoiding the up-and-down cycles of hemodialysis.

The dialysis prescription must be individualized for each patient. Generally, dextrose is used as the osmotic agent, with the concentration varying based on how much fluid must be removed. The higher the dextrose concentration, the more fluid will be removed—but more dextrose will also be absorbed, elevating blood sugar. Each dialysis exchange is generally two to three liters in volume. The dwell times and the number of exchanges per day vary depending on the patient and the characteristics of the peritoneal membrane.

There are several options for peritoneal dialysis. Continuous ambulatory peritoneal dialysis is performed manually four to five times during the day.

Figure 2. The Hemodialysis Process



During hemodialysis, blood is removed from the body through the tubing shown in red and is propelled along by a blood pump. The blood passes through the dialyzer and then is returned to the body through the tubing shown in gray. Along the way, blood pressure monitors, dialyzer inflow monitors, and air detectors help to ensure the patient's safety during the procedure. A heparin pump prevents the blood from clotting. Figures 2–4 are reprinted courtesy of the National Kidney Disease Education Program, a program of the National Institute of Diabetes and Digestive and Kidney Diseases.

Continuous cycling peritoneal dialysis involves the use of a cycler, a small appliance that performs the exchanges automatically. With the cycler, many patients can perform enough exchanges while asleep at night, such that they don't need additional exchanges during the day. Some patients need one or two additional manual exchanges during the day for adequate clearance. Most patients on peritoneal dialysis now use the cycler.

It's important for patients on peritoneal dialysis to restrict their potassium intake. (Such restriction may be greater for patients on hemodialysis.) Amino acids that are lost during the exchanges must be replaced, and the patient's dietary protein needs will be higher. Absorbed dextrose may cause the patient to gain weight. Because peritoneal dialysis is continuous, patients are never in a fasting state, and this has particular implications for those who have diabetes. Glucose levels may be harder to control, but insulin can be added to the dialysis solution. Some patients experience body-image concerns associated with the catheter, and may need psychological and emotional support.

Hemodialysis. In hemodialysis, patients are treated with a hemodialysis machine three or more times a week. A dialyzer serves as the filter, replacing the kidneys. The patient's blood is pumped from the body through tubing, passes through the dialyzer, and is returned to the body. Along the way, blood pressure monitors and airflow detectors ensure the patient's safety. (See Figure 2.)

The blood enters at the top of the dialyzer and is forced through multiple hollow filaments, each about the size of a human hair. Each filament acts as a semipermeable membrane. As blood passes through the filaments, the dialysis solution flows around the outside of the filaments. It takes less than one second for blood to pass from the top of the dialyzer to the bottom; as it does, waste products diffuse into the dialysate and are carried off, and the blood returns to the body. Diffusion efficiency depends on the size of the solute. Protein-bound substances usually aren't removed; some amino acids, glucose, and water-soluble vitamins are removed. (See Figure 3.)

In-center hemodialysis may be a choice for a patient who can travel to a dialysis center three times a week for scheduled treatments, prefers that trained staff handle the treatments, doesn't mind venipuncture, and is willing to follow a diet that includes numerous restrictions. Advantages of in-center hemodialysis include the availability of facilities nationwide and the presence of trained staff to do the work. If they so choose, patients can be relatively passive. The staff places the needles, monitors treatment, and maintains the equipment. As with many people with chronic illnesses, people with end-stage kidney disease may become socially isolated, and may enjoy the social setting of the dialysis center. Patients typically spend three to four hours three times a week with

the same relatively small group of other patients and providers. Disadvantages include more stringent dietary restrictions, the loss of nutrients during hemodialysis, limited control over the procedure, and the burden of travel to and from the center. Furthermore, hemodialysis patients never reach equilibrium, experiencing instead either a gradual increase in waste products and fluids between treatments or a rapid decrease of these during treatment. These up-and-down cycles may fatigue patients.

Home hemodialysis may be a choice for a patient who wants to perform in-home treatments, has someone to help in doing so, can perform treatments three or more times per week, has room for the machine and supplies, and doesn't mind needlesticks and self-cannulation.

Home hemodialysis is becoming more popular.[39] It requires training and support. As with in-center hemodialysis, home hemodialysis can be done three times per week. But it also permits other options, including daily dialysis for two to three hours, five to six times per week, and nocturnal dialysis for six to eight hours, three or more nights per week. More frequent home dialysis appears to be associated with significant benefits to the patient.[43]

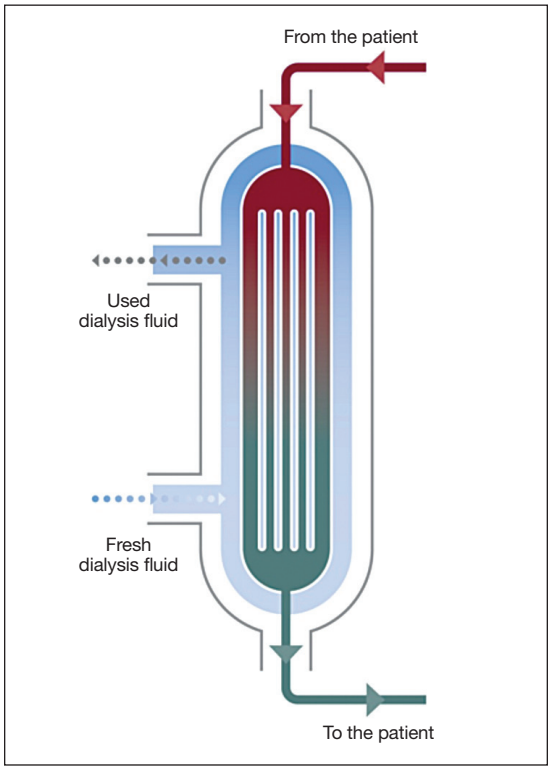
Home hemodialysis has a different set of advantages and disadvantages. On the one hand, patients have more control over their schedules, travel isn't required, and the newer machines are smaller and easier to use than the older models were. The diet may be less restrictive, and phosphate binders may be less necessary or not needed. And if treatments are done more frequently, the ups and downs are less severe. On the other hand, home hemodialysis requires that a second person (often a partner or other family member) be present to assist, which may cause stress to the patient, the other person, or both. Either the patient or the person assisting has to insert the needles; and the machine and supplies require space. The patient might have to take time off from work in order to get the initial training, which may not be offered locally. Protein requirements are higher because of protein losses during treatment.

Vascular access. To perform hemodialysis, vascular access must be created. In dialysis, blood usually flows at a rate of about 400 mL/min. Withdrawing blood at that rapid rate from any native peripheral vein would collapse that vein. A blood vessel that can withstand that withdrawal rate without collapse is required.

With permanent vascular access, an artificial connection between an artery and a vein is created, such that some blood is diverted to the vein. This connection may be direct or indirect. An *arteriovenous fistula* establishes a direct connection, and is the preferred access method, as it's less likely to become infected or to clot (see Figure 4A). Fistula maturation takes several weeks and involves dilation and thickening of the vein, which occurs as a result of increased blood flow. Once the fistula is mature, access to blood flow for dialysis occurs through a percutaneous needlestick. If a direct connection cannot be created because of small vessel size or another mechanical problem, then an *arteriovenous graft* is the second option. This connection is made indirectly, using a synthetic tube (see Figure 4B).

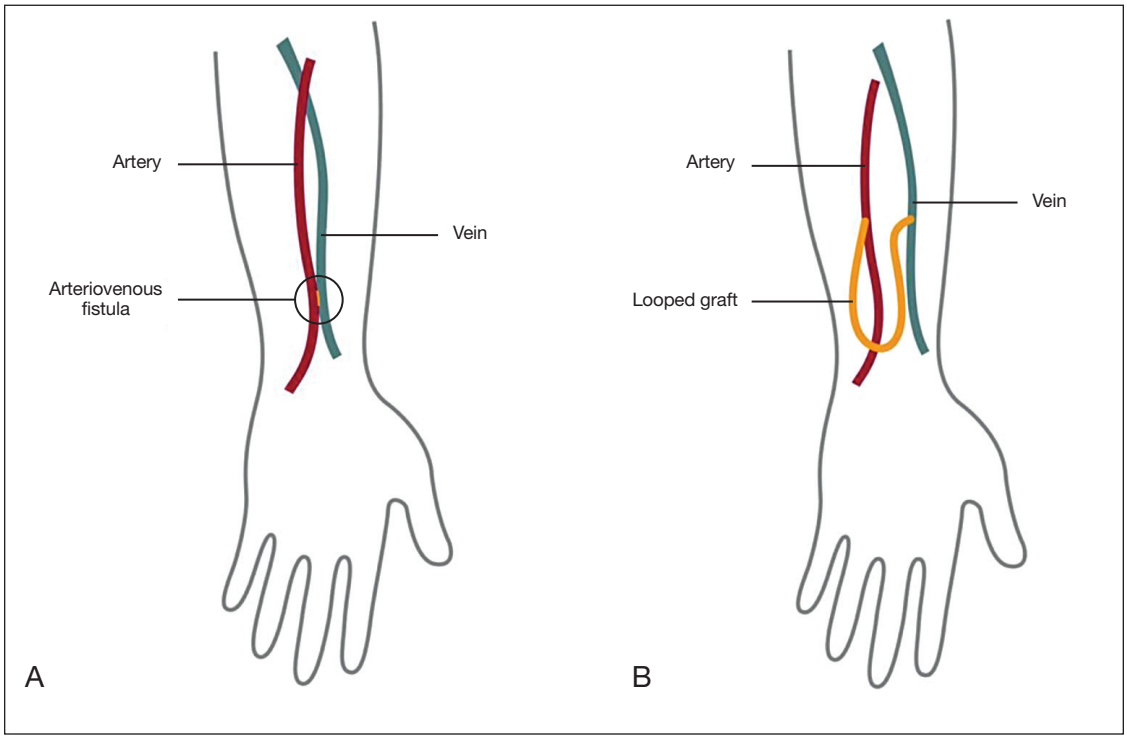
Permanent vascular access is usually established in the nondominant arm. A large IV line (such as a peripherally inserted central catheter) placed in a peripheral vein can destroy that vein for future dialysis use. Patients with CKD should be counseled to protect the blood vessels in both arms by avoiding venipuncture or IV catheter placement above the wrist, if possible. When emergent dialysis must be performed, temporary vascular access may be established using a central vein, usually by placing a catheter in the internal jugular vein in the neck. However, this is only a temporary solution. Catheters are associated with inadequate dialysis, increased infection rates, increased clotting, and inflammation.[44]

Figure 3. The Dialyzer Used in Hemodialysis



Blood enters at the top of the dialyzer and is forced through thin, hollow filaments made of semipermeable membrane. As blood passes through these filaments, the dialysis solution passes in the opposite direction on the outside of the filaments. Waste products diffuse from the blood into the dialysate. Filtered blood is returned to the patient.

Figure 4. The Arteriovenous Fistula and the Arteriovenous Graft



An arteriovenous fistula (A) establishes a direct connection between an artery and a vein. Once matured, the fistula allows access to blood flow for dialysis through a percutaneous needlestick. An arteriovenous graft (B) uses a synthetic tube to connect the artery and the vein. This method is used when a direct connection can't be established because of either small vessel size or another mechanical problem.

Supportive treatment without transplantation or dialysis. Opting for neither transplantation nor dialysis may be right for patients who feel that such treatments won't improve their health; feel they've done what they wanted to do in life; and, ideally, have family and friends who support their decision. Supportive treatment involves active medical management in which many complications can be treated. Medications for CKD are usually continued and can be adjusted. However, with supportive treatment there is no medical intervention aimed at replacing lost kidney function. Without clearance of uremic toxins, the patient will eventually become uremic.

It's essential to provide comfort and palliative care to these patients. Patients who choose supportive therapy need to understand that without kidney replacement therapy, they will eventually die from uremia; it's important that their family members understand this also. These facts must be presented in a manner that doesn't question the patient's decision, yet ensures that the decision is an informed one.

For additional information on caring for patients with CKD, visit the Web site of the National Kidney Disease Education Program (http://bit.ly/2gaGy4w). ▼

For eight additional continuing nursing education activities on topics related to kidney disease, go to www.nursingcenter.com/ce.

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