Chronic Renal Failure

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By Melissa K Slate, RN, BA, MA

Objectives

By the end of this lesson the health care professional will be able to:

Recognize the 5 stages of chronic kidney disease.

Explain the goals of treatment for each stage

Demonstrate knowledge of symptoms at each stage of progression

Describe common laboratory and diagnostic tests used in the management and treatment of CKD.

Introduction

Chronic kidney disease or failure describes the process by which the patient experiences a gradual decline in renal function regardless of the current stage of disease progression. These patients usually are not being treated with renal replacement therapy through dialysis or transplantation, although that need may be anticipated in the future. End stage renal disease (ESRD) is the is the term used to indicate the need for some type of renal replacement therapy is required by the patient in order to maintain survival.

Chronic renal failure is a continual and irreversible loss of kidney function over an extended period. Chronic renal failure has five stages based on the GFR (glomerular filtration rate) and patients may have no symptoms in the early stages. In chronic renal failure, the kidneys lose their ability to handle wastes. The body begins to lose the ability to handle water and to maintain chemical and metabolic processes. When kidney function decreases to 15% of normal dialysis or transplantation is required to maintain survival.

The kidneys remove excess wastes and by products from the blood. This accumulation of excess waste is called azotemia. The kidneys also regulate the fluid balance of the body, the failure of the kidneys to rid the body of excess water results in the accumulation of fluid called edema. Electrolyte balance is another function of the human kidney and alterations can result in hyper or hypokalemia, hyper or hypernatremia, and changes in the bicarbonate levels in the blood. Kidneys also regulate the production of red blood cells by erythropoiesis, which is the secretion of the hormone erythropoietin. The kidneys also regulate the production of bone, blood pressure, and acid-base balance.

Diabetes and High Blood Pressure are two very important risk factors for chronic renal failure, and their incidence is increasing. Age is also another risk factor for chronic renal failure due to the natural loss of nephrons, as we grow older. Having a family history of kidney disease or diabetes puts the patient at greater risk. The male gender is a risk for chronic renal failure due to the prevalence of males having chronic kidney disease. Also being from an ethnic background seems to pose a greater risk of chronic kidney disease as does smoking. Anemia and obesity play a role in risk factors for developing chronic kidney failure, as does the use of NSAIDS.

STAGE	GFR	SYMPTOMS
Stage 1	Kidney Damage GFR Normal or >90	Usually has no symptoms
Stage 2	Mild Kidney Damage GFR<60-89	Usually has no symptoms
Stage 3	Moderate Kidney Damage GFR < 30-59	Iron deficiency, anemia, hypertension, malnutrition, bone disease, and metabolic acidosis
Stage 4	Severe Kidney Damage GFR <15-29	Worsening of previous symptoms
Stage 5	Kidney Failure GFR <15	Worsen symptoms with difficulty thinking.

Goals of therapy

Stage 1

Diagnose and treat chronic renal failure
Treat other diseases present
Slow disease progression
Reduce risk of cardiovascular disease

In stage 1, the focus is on identifying the disease and starting appropriate treatment measures. It is important to define the etiology of the disease, as this will drive the treatment plan. In other words, if diabetes is present then treatment is driven toward tight control of blood sugar to prolong disease progression. Sometimes medications may directly treat the disease itself such as the use of steroids in Glomerulonephritis. Any cardiovascular disease that is present needs to be addressed as well, as persons with chronic renal failure are at increased risk of death from CV disease.

Interventions for slowing the progression of chronic kidney disease include; tight control of glucose in diabetic patients, strict control of blood pressure, and the use of Angiotensin- converting enzyme inhibitors. Diabetes is the number one cause of chronic renal failure in the United States. Hypertension ranks second. Both of these diseases damage the microvascular structure of the nephrons causing failure of the basement membrane. In diabetic patients, the goal is to keep the Hgb A1C to less than 7. Studies have shown an approximate 50% reduction in the progression of nephropathy occurs with tight glucose controls.

There are four stages of hypertension. Normal blood pressure is less than 120/80. Pre hypertension is defined as having a systolic reading of 120-139 and a diastolic reading of 80-89. Stage 1 hypertension is classified by a systolic blood pressure in the 140-159 range and the diastolic between 90-99. Stage 2 hypertension is a systolic pressure greater than 160 and a diastolic pressure over 100. If a patient's blood pressure falls between two categories, then the higher category is used for classification purposes. Controlling blood pressure has been shown to reduce the progression of chronic kidney disease.
The goal of hypertension therapy is a goal of blood pressure less than 140/90. Stricter control of blood pressure is recommended in patients with urine microalbuminuria or other evidence of diabetic kidney disease.

Modifiable risk factors are those that can be controlled or changed such as weight, salt intake, smoking, alcohol use, and sleep apnea, non-modifiable risk factors are those that cannot be changed such as age or heredity. Edema, which leads to elevated blood pressure, from excess fluid circulating in the blood, can be controlled with diuretics. Diet and exercise can be important tools for both modifying risk factor and contributing to a sense of well-being. Sleep apnea has

been associated with increased risk of adverse cardiac events, so symptoms of sleep apnea need to be assessed and addressed.

The first line of defense for diabetic patients needing hypertensive management should be ACE inhibitors or an angiotensin receptor blocker due to their renal protective effects. In fact, these medications can be started in advance of the patient showing hypertensive symptoms in an attempt to slow progression of chronic renal disease. Every attempt is made to maximize the dosages on these medications before new ones are added. The next drug to be added is a diuretic; loop diuretics are frequently utilized due to their superior performance in low glomerular filtration rates. Then beta-blockers or calcium channel blockers are added, however beta-blockers should be used cautiously due to their ability to mask hypoglycemia. Metoprolol is the preferred beta-blocker due to secretion by the liver instead of the kidneys.

In addition, the use of ACE inhibitors is recommended regardless of the hypertensive status due to the medication's ability to decrease glomerular blood pressure and protein filtration. ACE inhibitors should not be used in persons with bilateral renal artery stenosis, or in patients having renal artery stenosis and unilateral kidney. The rationale for this is that ACE inhibitors can cause marked decrease in renal blood flow in patients with renal artery stenosis. Any decrease in renal perfusion in a patient with compromised renal function can lead to further damage to the renal tissue from ischemia.

After the initiation of ACE inhibitors, assess the patient for side effects within 1-2 weeks of starting therapy and after any changes in dose. Up to 20% of patients may experience side effects from these medications. ACE inhibitors must be used with caution in dehydrated patients, sepsis, NSAID use, and renal artery stenosis due to the fact that these conditions can cause a superimposed acute renal failure. ACE inhibitors can harm the fetus so they must be discontinued if pregnancy should develop. ACE inhibitors can contribute to hyperkalemia, this may be managed with other interventions, and ACE inhibitors continued as long as potassium levels remain below 5.5 mg/dL. Sometimes a decline in GFR will occur with ACE inhibitors, monitor GFR, and stop medication if decline in GFR exceeds 30% of baseline for more than 4 months.

Treatment of hypertension in non-diabetic chronic renal disease patients is divided into two categories; those with urinary protein excretion and those without. For patients with proteinuria an ACE inhibitor or angiotensin receptor blocker is the first drug of choice, this again is due to the renoprotective effects. Next a diuretic is added and finally a beta-blocker or calcium channel blocker. The dose of each medication is maximized before a new drug is added. For patients without urinary protein excretion, the goal of blood pressure control is less than 125/75. Diuretics are utilized first, followed by an ACE inhibitor or an

angiotensin receptor blocker. Beta-blockers or calcium channel blockers are then added. It is not unusual to see a patient with chronic renal disease on several different medications for blood pressure control, five different medications is about the average.

All patients should be evaluated for elevated blood lipids using a complete fasting lipid profile. A trial of lifestyle management should be tried which includes diet and exercise as appropriate for the patient, and smoking cessation. If after 3 months the LDL remains in the 100-129 range, then drug therapy should begin with a statin medication. The goal for LDL is 70 mg/dl. If high triglycerides are present, then gemfibrozil should be the drug of choice as other fibrates may cause the creatinine level to rise.

Two major problems exist with statin medications. First, they can be toxic to the liver, so hepatic profiles should be monitored before the initiation of therapy and then at intervals as recommended for the medication. Another consideration is the association of statin medications with rhabdomyolysis. This condition is the breakdown of muscle tissue that accumulates in the kidneys and causes them to fail. This condition is potentially life threatening and should be considered if the patient complains of muscle pain after starting statins. If patients do not tolerate statins, then fibrates should be used.

There are numerous educational topics that should be covered with the chronic renal disease patient; so teaching needs to begin as soon as chronic kidney disease is identified. If the patient has family or other support system, they should be included in the teaching sessions if the patient is agreeable. The patient should be educated about renal anatomy, disease process, and progression; the patient should be taught about dietary measures and prevention of hyperkalemia, blood pressure control, medications, and lifestyle changes, and preparation for renal replacement modalities.

Stage 2

Treat chronic renal failure to slow progression
Treat cardiovascular disease
Determine the progression of the disease process

Estimation of the progression of the disease is important to evaluate interventions. Interventions may need to be re-evaluated as the disease progresses. Continued treatment of cardiovascular disease remains important because all patients with chronic renal failure are at risk for cardiovascular disease and should be considered in the highest risk group. Cardiovascular disease accounts for 40-50% of all mortality in end stage renal failure. Treatment

for cardiovascular disease includes risk assessment and reduction as well as specific therapies. Since the risk of cardiovascular disease rises as the GFR falls early intervention is essential.

Stage 3

In stage 3 of chronic kidney failure, the goals of treatment are the same as in stages 1 and 2 with the addition of the treatment of complications. By stage 3 chronic kidney failure, patients have a decrease glomerular filtration rate of 30-59 ml/min. and symptoms begin to develop. Evaluations for iron deficiency, anemia, hypertension, malnutrition, bone disease, and metabolic acidosis all need to be carried out at this stage as well as continuing evaluations for cardiovascular disease and dyslipidemia. Particular attention must be paid to medication dosages at this stage, as decreases in some medications need to be prescribed when GFR falls below 60ml per minute.

Symptoms of chronic kidney disease include loss of appetite and nausea due to the accumulation of excess waste in the body, which can lead to malnutrition. Edema and hypertension begin to occur. Headache, weakness, and insomnia may be present. Shortness of breath can be caused by either the resultant edema, from fluid accumulation, or from anemia. Itching is often accompanied by bone disease as phosphorus levels begin to rise in the blood and calcium is pulled from the bone. The itching is a direct result of phosphorus deposits in the skin.

Anemia is the loss of red blood cells below the level of 12g/dl in males and women past the age of menopause, and 11g/dl in females who are still menstruating. Anemia arises due to the inability of the kidney to produce erythropoietin, a hormone that stimulates the bone marrow to produce red blood cells. Iron deficiency is also common in stage 3 chronic renal disease and further enhances the anemia. Anemia can cause major impacts on quality of life.

Iron should be replaced if the ferritin level is less than 100ng/ml or the transferring level is less than 20%. Ferritin is the storage protein for iron in the body and is a good indicator of available iron stores. Iron replacement usually begins by prescribing oral iron supplements, if these are ineffective in raising iron levels, as may be the case in impaired iron absorption from the GI tract, IV iron preparations may be utilized. If these measures fail, then consideration should be given to beginning replacement erythropoietin therapy.

Erythropoietin replacement is available as erythropoietin alpha, which is dosed at intervals from once a week to three times a week. Darbepoetin alpha is a erythropoietin replacement with a longer half-life, it is dosed once weekly. With

each one of these medications, iron stores must be adequate before starting therapy. Therapy must be monitored and titrated to a Hgb of no more than 12. Contraindications for both medications are uncontrolled high blood pressure and sensitivity to any component of the medication.

Hemoglobulin is a more reliable indicator of anemia status that hematocrit. First Hgb is not influenced by a patient's fluid status. In dehydration, the hematocrit is falsely elevated or more concentrated due to the low volume state of the patient. With fluid overload, the hematocrit is falsely low due to dilution of the blood by water. Hgb is also stable when blood is stored at room temperature. Hct is calculated by this formula: MCV- erythrocyte count =Hct. MCV increases when blood is stored at room temperature, so the Hct calculation will be inaccurate. In addition, when blood glucose levels are elevated, as with many chronic renal disease patients, MCV will be falsely increased.

Anemia of chronic renal disease is normally normocytic and normochromic, meaning that the red blood cells are of normal size and shape. Microcytic cells, or cells that are abnormally small reflect iron deficiency, elevated aluminum, or hemoglobinopathies. Macrocytosis or abnormally large red blood cells can be associated with folate or Vitamin B12 deficiencies. Large red blood cells can also be associated with erythropoietin therapy as immature red blood cells enter circulation, and with iron excess. An elevated reticulocyte count can indicate a normal response to erythropoietin therapy, but can also indicate active blood loss as the reticulocyte count elevates in active blood loss. Abnormal white cell counts and platelet counts can indicate decrease bone marrow function.

Inflammatory conditions can impair normal erythropoietin production due to the presence of cytokines. Hypothyroidism can cause an anemia that closely resembles that of CKD; this is due to the slowing of the body processes from the decreased thyroid function. Osteitis fibrosa can cause anemia by replacing bone marrow with inactive tissue. Chronic blood loss (frequently from the GI tract) can deplete red blood cells and iron levels, sometimes faster than the body is able to compensate. Aluminum toxicity is not as common today as in the past with renal patients, but is still encountered in clinical practice. One source of aluminum is in certain antacid preparations. It affects the treatment length of anemia and the response to erythropoietin when the bone marrow is involved.

For adults, oral iron should be replaced at a level of 200mg per day. In pediatric patients, the dosage should be 2-3mg/kg. Oral iron replacement may not be adequate for adults due to multiple factors. Iron is best absorbed without food. If no response is noted from the administration of oral iron supplementation, then iron infusions should be started. Iron dextran can be given at 500-1000mg IV infusions after completion of a 25mg test dose and repeated as necessary. Annual iron loss in hemodialysis patients without other active blood loses can

attain levels of two grams and upwards. One milligram of iron is usually needed for each 1ml of packed red blood cells formed by the body.

When anemia is controlled, patients exhibit better quality of life and increased functional status. The target range for Hgb should be 11-13g/dl. Serum ferritin should be maintained at 150ng/dl and the transferring should be at least 20%. Poor outcomes are noted in patients with uncontrolled anemia. The have more hospitalizations than patients who are better controlled, their risk of death is greater, and their cognition is more impaired than their counterparts. In addition, anemic patients have greater difficulty completing self-care activities such as bathing and dressing due to shortness of breath and fatigue.

There are mainly two types of bone disease found in renal failure, osteitis fibrosa cystica and adynaminc bone disease. Osteitis fibrosa cystica is the most common type of bone disease found in chronic renal failure, and is characterized by high parathyroid levels (PTH). Adynamic bone disease is associated with normal or low levels of parathyroid hormone. Bone biopsy is the definitive test to differentiate between the two forms of bone disease. The body pulls calcium from the bone to the blood in an effort to control the rising phosphorus levels in the body. The relationship between calcium and phosphorus is inverse, when one goes up the other goes down.

When evaluating renal bone disease, the serum calcium, phosphorus, the calcium phosphorus product, the PTH level, alkaline phosphatase, vitamin D level, CO_2, and Aluminum level are drawn. Serum calcium, phosphorus, and PTH level provide the practitioner with direct information about bone status. The serum calcium and phosphorus should be normal and the PTH 35-70pmol/L in stage 3. The calcium phosphorus product is obtained by multiplying the calcium level times the phosphorus level, the normal result is 40. The treatment goal is for the result to be less than or equal to 55. Aluminum levels should be normal.

When the levels of PTH rise, reabsorption of phosphorus in the renal tubule is decreased, that is more phosphorus is excreted by the kidney. In chronic kidney disease, the kidney is unable to eliminate the phosphorus. Ionized calcium binds with phosphates, decreasing the levels of ionized calcium in the blood. This triggers increased PTH production and leads to a vicious cycle. When the calcium levels in the blood begin to fall, calcium is pulled from the bone in an attempt to meet the demands of the rising phosphorus levels. When hypercalcemia is resistant to other therapy, a parathyroidectomy is some times required to control the PTH level and regain control of calcium balance. High phosphorus levels in chronic kidney disease are treated with diet and phosphate binders. The patient needs to be educated about renal bone disease and encouraged to limit dietary forms of phosphorus. Dietary phosphorus can be found in nuts, beans, meat, dairy products, chocolate, eggs, and grains.

Phosphate binders are medications that bind dietary phosphate in the intestines so that it can be eliminated through the feces. Phosphate binders need to be taken with meals to be effective, and this needs to be stressed to the patient.

Vitamin D enhances the ability of the intestine to absorb calcium. When the kidneys fail, the ability of the body to convert vitamin D to a more active form is lost. This causes decreased serum ionized calcium levels to diminish and the levels of PTH increase. Replacing the active form of Vitamin D either orally or by IV enhances the intestinal absorption of calcium. The I.V. form of the medication seems to be slightly more effective. Vitamin D supplementation is most frequently given in conjunction with oral calcium. The calcium phosphorus product should be less than 55mg/dl when using vitamin D supplements.

Metabolic acidosis is the most frequently seen acid-base disorder in renal failure. The pH of the blood falls (becomes more acidic) due to impaired secretion of hydrogen ions. These ions are normally excreted by the kidney. In addition, the kidney loses the ability to reabsorb bicarbonate ions, which contributes to the alkaline qualities of the blood. The CO_2 level is diminished in metabolic acidosis. Metabolic acidosis leads to the breakdown of protein and can lead to malnutrition. It can also worsen bone disease as the body tries to buffer the excess acid in the body with calcium salts.

In metabolic acidosis, the goal of treatment is to keep the CO_2 level to between 20-22 mEq/L. The medication of choice is to use sodium bicarbonate. The usual starting dose is 0.5mEq/kg/day. One 650 mg tablet equals 7.7 mEq of bicarbonate. The patient should be educated about the importance of taking the medication and how to take it correctly. Sodium Bicarbonate cannot be used with some medications. Metabolic acidosis can cause myocardial depression and hypotension if not treated. Other effects included changes in mental status, coma, nausea, vomiting, and abdominal pain.

Protein malnutrition is directly correlated to morbidity and mortality among chronic renal disease patients. The mechanisms for this are many. Anorexia due to the accumulation of waste products can lead to decreased appetite, nausea, and vomiting. Often the patient will complain of alterations in taste that can be due to medication effects or metabolic processes. Also in renal disease protein can be excreted from the body faster than it is being restored, this is frequently seen in patients who are spilling large quantities of protein in their urine.

The treatment of protein malnutrition in chronic renal disease requires a multidisciplinary approach. One important intervention is referral to a renal dietician for nutritional counseling. Protein intakes less than 0.75mg/kg/day will actually worsen protein malnutrition. Metabolic acidosis must be adequately

controlled to manage protein catabolism. Nutritional supplements may need to be considered. The patient should be instructed on the addition of high biological protein sources, such as egg white, to the diet. Caloric intake of 35kcal/kg/day is required to prevent the breakdown of protein stores by the body to be used as energy. After dialysis is initiated, protein intake should exceed 1.2g/kg/day to offset the increased protein catabolism.

If the patient is not responding to treatment goals, the first thing to be considered is the patient's compliance, is he or she taking the medications as prescribed? If not then ask why. Financial issues may make it difficult for the patient to afford medications. It is not uncommon for patients to ration their medications or to run out of them before their next payday. Does the patient understand what the medications are for and the importance of taking them? Is the patient having side effects from the medication? Is the patient using OTC or herbal medications, or illegal drugs that may interfere with therapy? Also, evaluate the patient for potential decline in their renal function.

Stage 4

The primary goal is to treat complications and prepare for renal replacement therapy.

Patients who have been diagnosed with chronic renal disease will progress through several different stages of the grieving process. At first, the patient may experience denial and isolation in response to the diagnosis. Feelings of anger may be experienced as the patient begins to ask "Why me?' The bargaining stage is evidenced when the patient begins to feel that certain behaviors will change the outcome of the illness. Depression may begin to form as the patient begins to acknowledge the reality of his or her situation. Finally, the acceptance stage is attained, when the patient comes to terms with the diagnosis.

Stage 4 CKD is also described as severe. Most complications will be present at this level. In addition, volume overload and hyperkalemia are more prevalent at this stage. Preparation for renal replacement therapy should include dialysis options and transplantation as well as vein mapping and referral for access when appropriate. The patient and health care team should choose the placement of an AV fistula over an AV graft whenever possible. Long-term use of a central venous catheter should be avoided as it is the least desirable access for longterm use and has the greatest amount of complications. Follow-up appointments will be more frequent to control complications and symptoms.

Patient education that was initiated in stage 3 should be reinforced and increased during stage 4. Patients need to understand their disease process and

treatments to be empowered for self-management. Teaching about treatment modalities should be conducted and a choice made. The modality will determine whether the patient needs placement of a vascular access or a peritoneal dialysis catheter. Patients also need to know that they have the right to choose no treatment as an option; but be well advised that this choice of treatment will result in death. If the patient chooses no treatment, proper referrals need to be made to hospice, clergy, and support resources, ect., to assist the patient and family in making the dying process as dignified and as comfortable as possible when stage 5 is attained. In stage four, when the patient chooses no treatment, the goal is to make the patient as symptom free and comfortable as possible.

Stage 5

The Goals are to treat complications, initiate dialysis, and/or provide palliative care.

More frequent follow up will be necessary at this stage until the patient begins dialysis. Hyperkalemia, volume overload, uremia, and nutritional status will all have to be carefully monitored. Ace inhibitors may need to be stopped at this point, if they haven't already been, due to hyperkalemia. If the patient is having little appetite, or is having nausea and vomiting, careful assessment of fluid status will be vital because patients can loose muscle mass and replace it with fluid without appearing to loose any weight. Be especially careful if the patient doesn't seem to have weight changes, it may be fluid not body weight. Patients at this stage may also be sleepy or exhibit slowed thinking, so instructions will need to be written as well as verbally spoken. If possible, inform family members, so they may assist the patient if instructions are forgotten.

Diagnostic Tests for Chronic Renal Disease

Recommended screening tests for chronic kidney disease are: evaluation of creatinine, evaluation of blood pressure in both sitting and standing positions at every patient encounter, evaluation of cardiovascular risk, auscultation of heart and lungs, urine for protein, and assessment for end organ damage. It is also important to review a 12 lead EKG and palpate pulses. Urine protein can be a marker for kidney damage, so it is essential that it be evaluated on a regular basis. Manifestations of end organ damage can include absent pulses indicating peripheral vascular disease, retinal hemorrhages, history of stroke or TIA, or abnormal echocardiograms. Evaluation of kidney function should not be based solely on the serum creatinine. Creatinine levels are higher in men, those younger in age, and African Americans. Persons with higher muscle mass will have higher levels of creatinine.

Urine dipstick is a rapid screening tool that is performed to detect protein, bacteria, red blood cells, eosinophils, neutrophils, and bacteria. Urinary sediment examinations are necessary to detect specific types of kidney conditions through the presence of tubular epithelial cells, casts, crystals, fungus, and parasites. Urinary tests for proteinuria/microalbuminuria are one of the earliest indicators of kidney damage. Microalbumin can be detected in the urine long before tests are positive for total protein. An abnormal level of albumin is defined as greater than 30mg/ 24 hours. Abnormal levels of total proteins are defined as greater than 300mg /24 hours.

The presence of protein in the urine can be a valuable clue to the differential diagnosis and type of kidney disease present. Urinary protein is usually found in diabetic kidney disease, and glomerular diseases of both native and transplanted kidneys. Urinary protein is usually not associated with cystic kidney disease, vascular diseases of the kidney, and tubulointerstitial disease. Increasing amounts of urinary protein is associated with rapid declines in kidney function and cardiovascular disease.

Ultrasound of the kidney is one of the most useful, noninvasive tests for evaluating the appearance of the kidneys. Ultrasound can show the size, and shape of the kidney. Echogenicity, or the ability to produce echoes, indicates cystic kidney disease or medical renal disease. Small kidneys may indicate advanced renal disease, due to tissue shrinkage with advanced progression; large kidneys can indicate tumors, nephrotic disease, or infiltrations. Doppler exams of the kidneys give information about the status of blood flow to the kidneys and are useful in detecting renal artery stenosis. IVP tests can show the size, and shape of the kidney, as well as scarring, stones, and tumors; however, this test involves the use of contrast dye which may further damage compromised kidney function and is not often used in renal failure. CT and MRI scans can also be used to show structure and physical characteristics as well as detect cysts, stones, and renal artery stenosis.

Hemoglobin and Hematocrit are performed to measure the total and percentage of red blood cells respectively. Hgb is considered the more accurate indicator as it is not affected by the patient's fluid status. Reticulocytes are immature red blood cells and reflect the rate at which the bone marrow is producing new RBCs. Iron, ferritin, total iron binding capacity, and transferring saturation are all measures of iron utilization by the body. B12 and folate levels are measured, as deficiencies are known to contribute to and worsen anemia. Stool for occult blood is performed to check for active blood losses via the GI tract. PTH is performed because hyperparathyroidism interferes with erythropoiesis.

Conclusion

Chronic kidney disease is a major medical problem within the United States. The rates of kidney disease have more than doubled during the last 15 years. At risk persons should be identified, and screened for CKD at each patient encounter. Once a patient has reached Stage 3 of CKD, a nephrologist consult is vital to assist in preventing disease progression. Early referral decreases mortality and morbidity in the CKD population.

Hopefully, with earlier screenings and improved teaching targeted at chronic renal disease, fewer patients will be spending less time on dialysis. Nurses can play a pivotal role in stemming this medical epidemic through their educational efforts.

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