CHAPTER 6
Chronic Kidney Disease

6.1 Etiology of Chronic Kidney Disease in Rural Rwanda

There are no published population-based studies of kidney disease in Rwanda. Studies from other countries in sub-Saharan Africa suggest that most renal failure is probably caused by infections and hypertension. The major infectious causes are probably streptococcus, malaria, and tuberculosis (see **TABLE 6.1**).[1] Untreated urinary tract infections can also lead to renal dysfunction. HIV nephropathy is a major cause of kidney disease in areas with high HIV prevalence. Schistosomiasis may cause renal failure by obstructing the ureters. Other chronic diseases such as heart failure can also cause chronic renal failure. Toxins, such as those from a snakebite or from traditional medications, can directly injure the kidneys.[2] Patients who are seriously ill from any cause will often develop renal failure because of poor perfusion of the kidneys. Diabetic nephropathy is relatively rare in sub-Saharan Africa.

By contrast, the causes of acute renal failure are similar throughout the world. They are most frequently dehydration, infection, hypotension, and exposure to nephrotoxins.

Sub-Saharan Africa has a relatively low estimated prevalence of end-stage renal disease: 100 cases per 1 million people, compared with 1500 cases per million in the United States and 400 cases per million in Latin America.[2] By this measure, a country such as Rwanda, with a population of approximately 10 million, would have about 1000 people who require renal replacement therapy. This low prevalence reflects both the relatively high mortality associated with renal failure in sub-Saharan Africa and also the relatively low rate of diabetes and hypertension, the major drivers of renal failure in more developed countries.

TABLE 6.1 Causes of End-Stage Renal Disease in Sub-Saharan Africa and the U.S.

	Glomerulonephritis or unknown	Hypertension	Diabetes	Other
Average (Nigeria and Senegal)[1]	54%	27.4%	11.9%	6.7%
USA[3]	15.4%	24.2%	37.3%	22.9%

6.2 Screening for Renal Failure in High-Risk Populations

Most patients followed for renal failure in the Rwandan NCD clinics presented with overt symptoms such as anasarca. These patients had typically been hospitalized and then referred to the clinic at discharge. Many had end-stage renal disease. Access to renal replacement therapy or renal transplantation is extremely limited in Rwanda.

ACE inhibitors given to patients early in the course of their disease can dramatically retard the progression of kidney damage. Accordingly, the International Society of Nephrology has suggested population screening for urine protein as part of an integrated, community-based, case-finding strategy.[4,5] Some sites in resource-poor settings, including sub-Saharan Africa, have begun to investigate the value of this approach.[6] In Rwanda, we have not yet pursued population screening. Instead, we have focused initially on improving the care of patients with advanced disease (including palliation) while screening for renal disease in high-risk populations.

The three populations identified as at risk for asymptomatic chronic renal disease in Rwanda are those with hypertension, diabetes, and HIV. These patients are followed in their respective continuity clinics (at health-center level). Referral to the district-hospital NCD clinics is triggered once a creatinine test is needed.

6.2.1 Hypertension

All adult patients with a persistent blood pressure greater than 160/100 mmHg are screened for proteinuria. Patients found to have greater than 2+ proteinuria in an uncontaminated specimen are referred to the district NCD clinic for confirmation of proteinuria, and to have their creatinine measured (see **SECTION 8.2** and **PROTOCOL 8.2**). In addition, adult patients younger than 40 who have stage II or greater hypertension are referred to the district NCD clinic for creatinine measurement (see **PROTOCOL 8.3** and **SECTION 8.6**). Hypertension in these patients may be secondary to another cause; advanced renal disease is one common culprit. Hypertension in children ≤ 15 years is almost always a sign of an underlying disorder, most frequently renal failure. These patients are referred to the district hospital for an inpatient evaluation, including assessment of renal function (see **SECTION 8.8**).

6.2.2 Diabetes

All patients diagnosed with diabetes should have a urine dipstick test performed twice per year and serum creatinine checked annually. Urine microalbumin testing is currently not available.

6.2.3 HIV

HIV is thought to cause renal failure primarily through damage to the glomerulus, resulting in HIV-related nephropathy (HIVAN).[7,8] Studies in sub-Saharan Africa report HIVAN prevalence that varies by population and severity of HIV disease, ranging from 6% to 45% of HIV patients. Higher rates are associated with more severe HIV disease and hospitalization at the time of survey. Most studies show an improvement in renal function with initiation of highly active antiretroviral therapy, or HAART.[7]

We recommend that all HIV patients be screened for proteinuria. Those with proteinuria should be started on an ACE inhibitor unless contraindications exist. The WHO categorizes all HIV patients with symptomatic HIV-associated nephropathy as meeting the definition of stage IV HIV disease, regardless of CD4 count. Patients with HIV and nephrotic syndrome or kidney failure should be started on antiretroviral therapy at any CD4 count. Some experts have advocated that all HIV patients with proteinuria and no other clear cause should be started on antiretrovirals, especially in places where renal replacement therapy is unavailable. This may be difficult in settings where many patients with low CD4 counts are still not on treatment because of lack of resources. Some antiretrovirals, such as tenofovir, should be dosed differently or not used in renal failure.

In screening for renal failure with a urine dipstick, it is very important to avoid false positives. **TABLE 6.2** shows the approximate relationship between urine dipstick results and 24-hour urine protein collection. Urine concentration affects dipstick results. For this reason, we have chosen a threshold of 2+ positivity. This threshold avoids false positives because it corresponds to more than 500 mg of protein in 24 hours at almost any urinary concentration. An alternative approach is to base the urine dipstick threshold on the specific gravity.[9] There is an online calculator that assists with this as well (see **http://www.metrohealthresearch.org/schelling**).

TABLE 6.2 Relationship between Urine Dipstick and 24-hour Urine Protein Results

Urine dipstick result	24-hour urine protein	Dipstick result may be suggestive of:
Trace	150 mg	Top-normal
1+	200–500 mg	Microalbuminuria*
2+	0.5–1.5 gm	Proteinuria
3+	2–5 gm	Nephrotic-range
4+	7 gm	Nephrotic-range

*Most urine dipsticks are insensitive for microalbuminuria

If initially positive, urine dipsticks should be repeated to confirm the result on at least 2 out of 3 tests. Proteinuria often accompanies urinary tract infections, but a positive test can also be caused by perineal cells contaminating the specimen. In practice, we recommend that any urine with epithelial cells, leukocytes, or nitrites not be regarded as positive for proteinuria. If there are many epithelial cells, the test should be repeated on a fresh specimen, collected using clean catch techniques. Young children may require cathertization to obtain a clean catch specimen. If there are many leukocytes (≥ 2+) but not many epithelial cells, the clinician should consider treating the patient for a urinary tract infection if there are clinical reasons to suspect one.

6.3 Classification of Renal Failure

Renal failure severity classification is traditionally based on interpretation of the serum creatinine in combination with the patient's weight, sex, and age. These factors combine to give a glomerular filtration rate (GFR), which is an estimate of how well the kidneys filter the blood. A high GFR means the kidneys are filtering the blood appropriately, while a low GFR means the kidneys have lost the ability to filter the blood very well. The formula most commonly used worldwide to estimate GFR is one developed by Donald Cockroft and Henry Gault in the 1970s in Canada (**http://www.nephron.com/cgi-bin/CGSIdefault.cgi**).[10] A report from Ghana has shown that the Cockroft-Gault equation tends to significantly overestimate the degree of kidney failure in the studied population.[11] An alternative, known as the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, estimates renal function more accurately in individuals with normal renal function and has been validated in Ghana.[12] This equation is difficult to use without an online calculator (**http://www.kidney.org/professionals/kdoqi/gfr_calculator.cfm**). Given concerns about overdiagnosis, we do not recommend using Cockroft-Gault in asymptomatic patients being screened

for kidney disease.[13] There is also an online calculator available for the Schwartz equation for use in pediatric populations under age 15 (**http://www.kidney.org/professionals/kdoqi/gfr_calculatorPed.cfm** or **http://nephron.com/bedside_peds_nic.cgi**).[14]

An initial injury to the kidney can cause damage to the renal parenchyma. The kidney responds by hyperfiltration, forcing the remaining normal nephrons to work harder to maintain the same clearance of toxins from the blood. Therefore, in early chronic kidney disease, the creatinine initially does not rise. However, proteinuria may be present, suggesting some dysfunction of the nephrons that allows protein to escape from the blood to the urine. Initiation of treatment with ACE inhibitors to decrease hyperfiltration can help to protect the kidneys from further damage.

Calculating GFR for each patient in a busy clinic may be impractical. For this reason, we often give absolute cutoffs of creatinine to guide diagnosis and treatment decisions. These cutoffs are purposefully conservative to account for our patients' low average body weight and for the difficulties of closely monitoring electrolytes in our setting.

TABLE 6.3 outlines the different stages of chronic kidney disease (CKD) with corresponding ranges of GFR and creatinine for adults and children. These creatinine ranges are purposefully conservative because many of patients have low body weight. **TABLE 6.4** lists the average creatinine for age for children. The table refers to the glomerular filtration rate. CKD stages 1 and 2 refer to patients with normal GFRs (≥ 60 ml/min), but with some degree of proteinuria (including microalbuminuria). Patients with CKD 3 have significant kidney impairment but are usually asymptomatic. Patients with CKD 4 and 5 have severe kidney dysfunction. These patients are likely to begin experiencing symptoms. **TABLE 6.4** gives corresponding levels of creatinine elevation for an average patient in our clinic.

TABLE 6.3 Stages of Chronic Kidney Disease

	Degree of dysfunction	Glomerular filtration rate	Approximate creatinine cutoff for adults	Approximate creatinine cutoff for children
CKD 1 and 2	Mild dysfunction	≥ 60 ml/min/1.73m^2	< 100 µmol/L (< 1.1 mg/dL)	Normal creatinine for age
CKD 3	Moderate dysfunction	30–59 ml/min/1.73m^2	100–199 µmol/L (1.1–2.3 mg/dL)	Normal to < 2x normal creatinine for age
CKD 4 and 5	Severe dysfunction	≤ 29 ml/min/1.73m^2	≥ 200* µmol/L (> 2.3 mg/dL)	≥ 2x normal creatinine for age

* Conservative lower limit; corresponds to a GFR of about 30 ml/min in a 30-year-old, 50 kg woman.

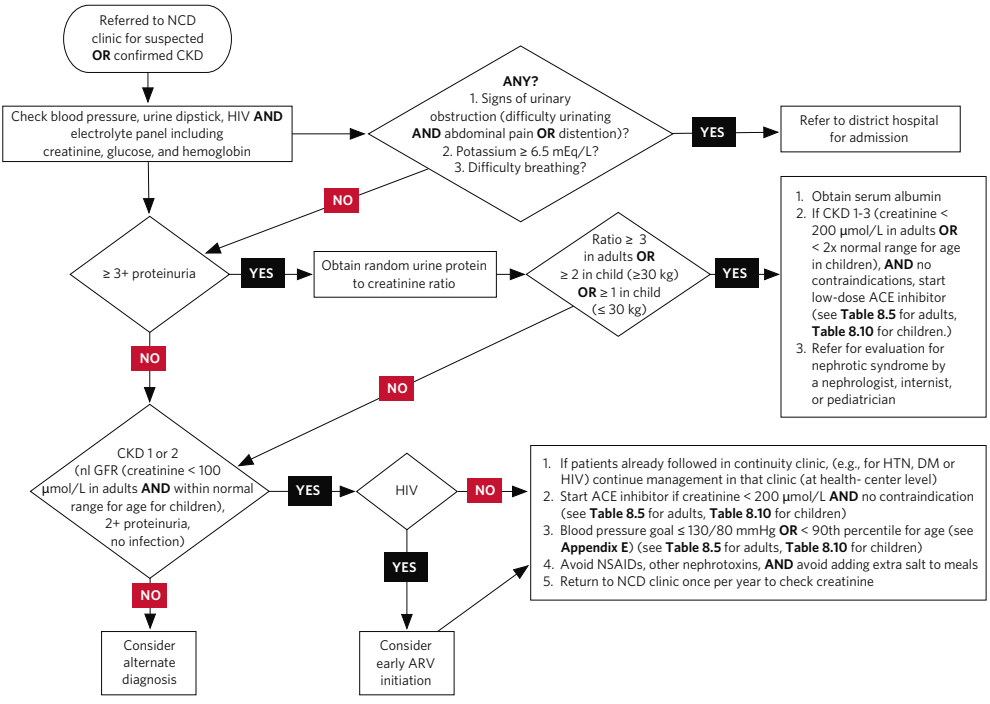
TABLE 6.4 Normal Creatinine Ranges for Children by Age

Newborn	27–88 µmol/L (0.3–1.0 mg/dL)
Infant or pre-school-aged child (2 months–4 years)	18–35 µmol/L (0.2–0.5 mg/dL)
School-aged child (5–10 years)	27–62 µmol/L (0.3–0.7 mg/dL)
Older child or adolescent (>10 years)	44–88 µmol/L (0.5–1.0 mg/dL)

6.4 Initial Evaluation and Management of Chronic Kidney Disease (CKD)

PROTOCOL 6.1 and **PROTOCOL 6.2** outline our approach to the initial evaluation of kidney disease based on the severity of dysfunction. Most individuals are found to have CKD 1 or 2 through dipstick screening after they are already diagnosed with HIV, diabetes, or hypertension. These patients are referred to district-hospital NCD clinics to have their creatinine evaluated, since creatinine testing is not usually available at health centers. If there are signs of urinary obstruction or difficulty in breathing, patients should be hospitalized for further evaluation and treatment. All patients should have the following tests on their initial visit to the NCD clinic: blood pressure, urine dipstick, HIV test, electrolyte panel, creatinine, glucose, and hemoglobin.

PROTOCOL 6.1 Initial Evaluation of Chronic Kidney Disease Stage 1 or 2



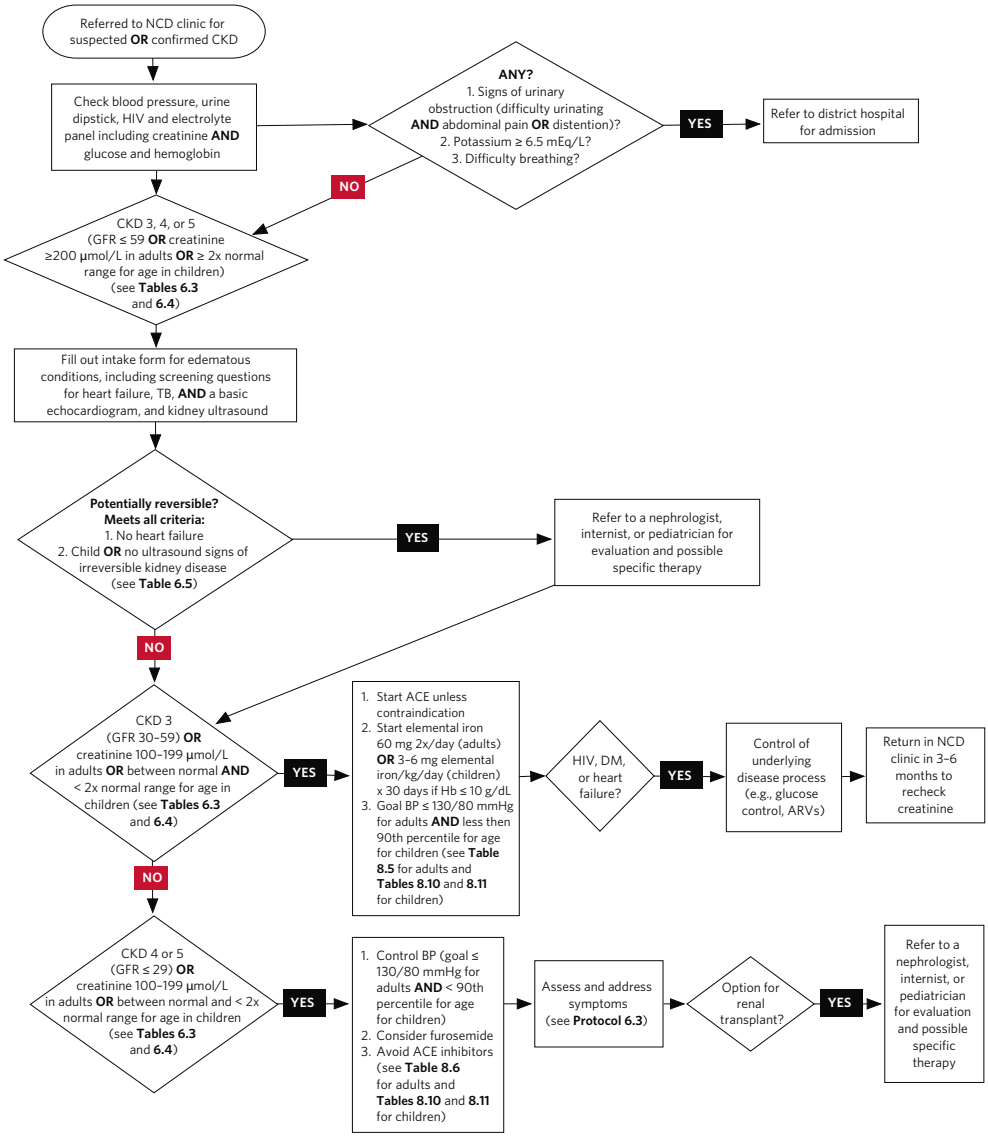
If there is evidence of 3+ or 4+ proteinuria on the urine dipstick, a spot urine protein–to-creatinine ratio should be obtained. The spot urine protein–to-creatinine ratio (mg per mg) correlates well with the number of grams of protein excreted in a 24-hour period. This will determine if there is nephrotic-range proteinuria (more than 3 gm in 24 hours in an adult or 50 mg/kg/day for children). For example, if a patient has 350 mg/dL of protein on a random urine sample and 50 mg/dL of creatinine, the urine protein–to-creatinine ratio would be 7. This corresponds to roughly 7 gm of urine protein output in 24 hours. It is important to use the same units for both protein and creatinine.

Patients with nephrotic-range proteinuria should be referred to the district or referral center level for consultation by either a nephrologist (if available), pediatrician, or internist for evaluation and initiation of steroids or other therapy if needed. It is also helpful to obtain a serum albumin test prior to further evaluation. If the patient has mild to moderate renal dysfunction (CKD 1–3, see **TABLE 6.3** and **TABLE 6.4**), it is helpful to start a low dose of an ACE inhibitor (see **TABLE 8.5** for adults, and **TABLE 8.10** and **TABLE 8.11** for children).

Once the diagnosis of CKD 1 or 2 is confirmed, patients should be followed at their usual health center clinic site. An ACE inhibitor should be initiated at a low dose (see **TABLE 8.5** for adults, and **TABLE 8.10** and **TABLE 8.11** for children) with a goal blood pressure of less than 130/80 mmHg in adults and ≤ 90th percentile for age for children (see **APPENDIX E**). Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, should be avoided along with other kidney toxins. As discussed above, HIV-positive patients should be considered for anti-retroviral therapy regardless of CD4 count to prevent progression of disease. Patients with CKD 1 or 2 should be seen once a year in a district-level NCD clinic to have their creatinine checked and their degree of CKD reassessed.

Patients with moderate to severe CKD (CKD 3, 4, or 5) should be closely evaluated for reversible causes of their disease (see **PROTOCOL 6.2**). Patients with CKD 4 or 5 will often be symptomatic and initial evaluation will take place in the hospital. Patients with CKD 3 are usually identified through screening because they have another disease (such as heart failure).

PROTOCOL 6.2 Initial Evaluation of Chronic Kidney Disease Stage 3, 4, and 5



When there is no obvious cause of kidney dysfunction, the key question is whether the patient has a condition that would benefit from treatment with steroids or another immune-modifying agent. At the district hospital level, ultrasound is one simple tool that can help identify patients unlikely to benefit from specific therapies.

The *PIH Manual of Ultrasound for Resource-Limited Settings* describes how to obtain and interpret ultrasound images of the kidneys.[15] The guide is aimed at district clinicians. Assessment for kidney size and the

presence of hydronephrosis should be part of the training of generalist physicians, clinical officers, and NCD nurses. Normal kidneys in an adult measure 9–13 cm in length and 4–6 cm in width. Additionally, the kidney cortex (outer portion of the organ) is usually relatively dark, compared to structures like the liver on ultrasound (see **FIGURE 6.1** and **FIGURE 6.2**). Most sources of renal disease will cause the kidneys to become smaller and brighter. Important exceptions are HIV and diabetic nephropathy (conditions that are not thought to be particularly steroid-responsive).

FIGURE 6.1 **Normal Kidney Anatomy on Ultrasound** ***(Arrowheads show the medullary pyramids, and star shows the outer cortex)***

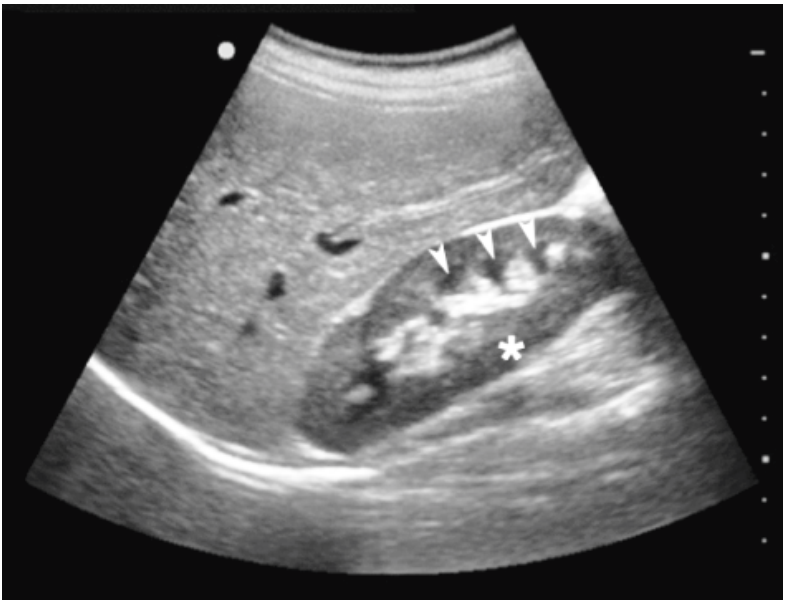
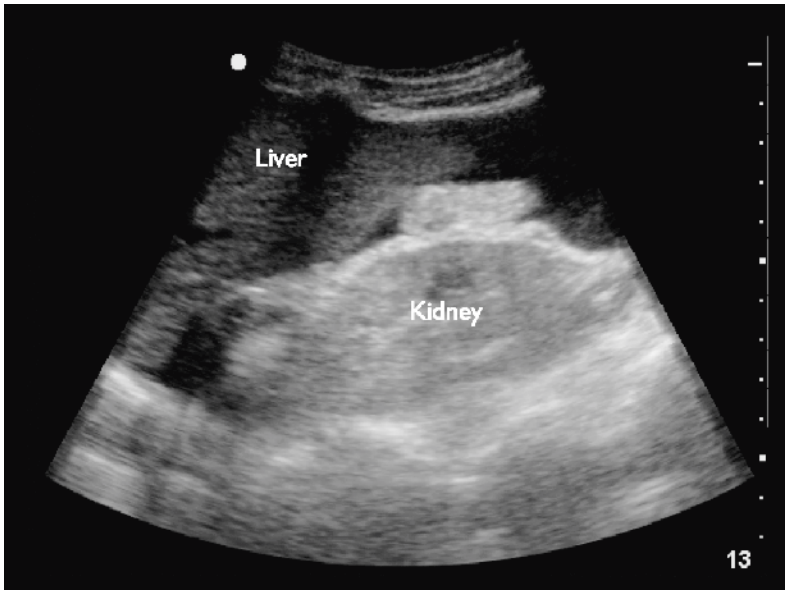


FIGURE 6.2 Ultrasound in Chronic Kidney Disease: Loss of Differentiation between Cortex and Medulla



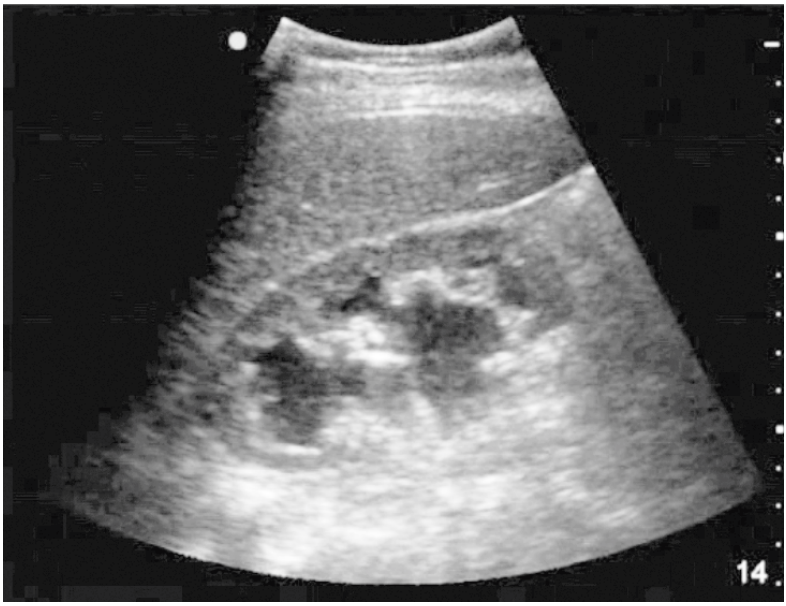
In general, kidneys that are very small and echogenic (bright on ultrasound) are likely to be affected by advanced, chronic disease that will not respond to steroids or other specific therapies. A recent study has correlated ultrasound and renal biopsy results.[16] This study found advanced, irreversible disease in 86% of patients who had both small kidneys (≤ 20 cm combined length in adults), and echogenic cortices (brighter than the liver) on ultrasound. Echogenicity alone was fairly non-specific (only 30% had advanced disease). The following combination of findings on ultrasound should preclude the need for specialist referral (see **TABLE 6.5**). Normal kidney size in children will vary by age. However, children are more likely than adults to have reversible renal failure and should always be referred to a nephrologist or pediatrician for further evaluation, regardless of ultrasound findings.

TABLE 6.5 Ultrasound Findings Specific for Severe, Irreversible Kidney Disease

1. Echogenic cortex (more than liver), plus a combined length ≤ 20 cm in adults **OR** ≤ 2 standard deviations below normal for age in children
2. Very small kidneys with a combined length less than 16 cm in adults

Obstructive nephropathy is a potentially reversible cause of renal failure that should prompt hospital admission for further evaluation (see **FIGURE 6.3**).

FIGURE 6.3 Hydronephrosis on Ultrasound



In this chapter we do not go into the details of evaluation by a nephrologist. In many settings, renal biopsy is not available. Decisions to start steroids or other therapies may have to be made with limited data. Although availability of renal replacement therapy is very limited, it may play a role in patients with acute and potentially reversible disease. Issues surrounding renal transplantation are discussed below.

The roles of the district clinician are (1) to avoid referrals for patients unlikely to be candidates for specific therapies; and (2) to direct the ongoing care and palliation of such patients.

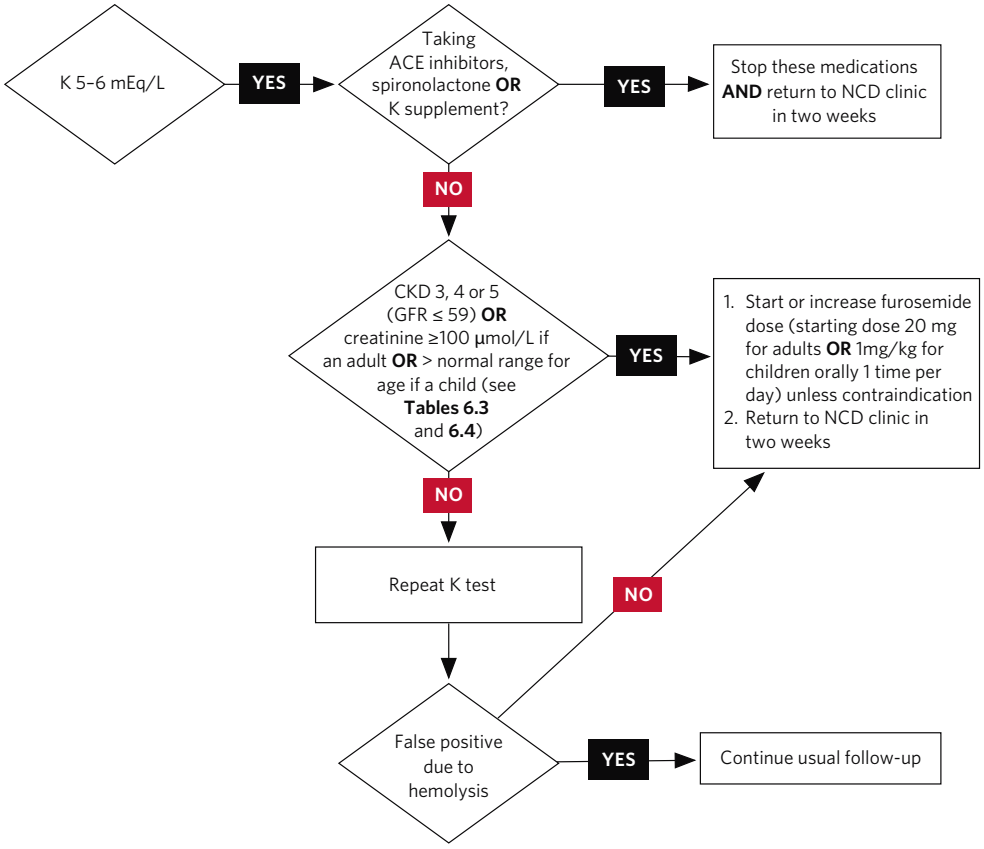
All patients with moderate renal failure (CKD 3) should receive iron supplementation if their hemoglobin is less than 10 mg/dL. The preferred dose for adults is 60 mg twice per day of elemental iron for 30 days. The dose for children is 3–6 mg elemental iron/kg per day. Other adjuvant therapies such as phosphate binders, calcium or sodium bicarbonate are not helpful unless patients are candidates for renal replacement therapy. Additionally, blood pressures should be kept below 130/80 mmHg (for adults) and below the 90th percentile for age (for children) if possible. Patients with CKD 3 (usually with a creatinine between 100 and 200 μmol/L in adults or between normal and 2x normal range for age in children) should still be able to tolerate and will benefit from ACE-inhibition (see **TABLE 8.5** for adults, and **TABLE 8.10** and **TABLE 8.11** for children). Contraindications include a potassium greater than 5 mEq/L. Furosemide is often a helpful adjuvant therapy.

The management of patients with severe renal failure (CKD 4 or 5) without a reversible cause for their disease depends upon the availability of renal transplantation. All patients should have their blood pressure controlled if possible (see **TABLE 8.6** for adults and **TABLE 8.11** for children). ACE inhibitors should be avoided. High doses of furosemide may be required. Patients should be assessed for symptoms of their disease (see **SECTION 6.6**). These symptoms should always be addressed. When symptoms become a predominant concern and if there is no access to life-prolonging therapies such as renal replacement or transplantation, the focus should be on quality of life.

6.5 Hyperkalemia

Potassium testing has become increasingly available at district hospitals in Rwanda. All patients evaluated for CKD 3 or higher at district NCD clinics should have their potassium checked initially. If a patient is found to have hyperkalemia (≥ 5 mEq/L), ACE inhibitors, spironolactone and any potassium supplementation should be discontinued (see **PROTOCOL 6.3**). If there is an abnormally high potassium level in a patient with an estimated glomerular filtration rate of more than 60 mg/ml, in the absence of culprit medications, a false reading based on hemolysis should be suspected.

PROTOCOL 6.3 Outpatient Management of Mild Hyperkalemia



In patients with mild elevations in serum potassium (5–6 mEq/L), several medications can help with the excretion of electrolytes. Furosemide is an effective potassium-wasting diuretic. If the patient does not have heart failure, the dose of this medication should be increased carefully until potassium levels normalize. The patient should be followed every two weeks to monitor for signs of hypovolemia.

Patients with a confirmed K ≥ 6 mEq/dL should be hospitalized to receive treatment that could immediately decrease potassium levels. We do not include ECG testing routinely in this management protocol. Acute therapies include intravenous insulin administered along with glucose, intravenous calcium gluconate, and albuterol. Chronic therapies include furosemide and albuterol. Cation exchange resins such as sodium polystyrene sulfonate are not readily available. We do not discuss hospital management of hyperkalemia in this section. We refer the practitioner to the forthcoming WHO district clinician manual for adult and adolescent medicine.

If the patient has hyperkalemia at baseline in the setting of severe, irreversible renal dysfunction (CKD 4 or 5), the decision about the intensity of therapy should depend on discussion with the patient about the goals of care.

6.6 Palliative Care for Chronic Kidney Disease

Palliative care entails responding to and relieving suffering of any kind, whether physical, psychological, social, or spiritual (see **CHAPTER 2**). PIH's experience in responding successfully to social suffering with social supports—including housing, nutrition, clean water, and daily visits by community health workers—are described in the *PIH Guide to Community-Based Treatment of HIV in Resource-Poor Settings*.[17] This section will focus on the physical and psychological effects specific to advanced chronic kidney disease (CKD).

There is no contradiction between treatment of CKD and palliative care. Every effort should be made to prevent and slow the progression of early-stage chronic kidney disease. At any stage of CKD, pain, dyspnea, and other distressing symptoms should be treated. In the later stages of CKD, many patients will have an increasing need for palliative care, including relief of physical symptoms and psychosocial distress.

The number and severity of physical and psychological symptoms experienced by patients with CKD 5 is similar to those experienced by patients with advanced cancers and other chronic, life-threatening illnesses. Symptoms of patients with CKD 5, their frequency, their known or presumed causes, and recommended treatments are listed in **TABLE 6.6** and **TABLE 6.7**.

TABLE 6.6 Common Physical Symptoms in Advanced Chronic Kidney Disease and Their Treatment[18]

Physical symptom	Cause	Treatment (see CHAPTER 2 for details)
Fatigue and drowsiness 86%	Depression	Assess for depression and treat if found.
	Fluid overload	Diurese for fluid overload, if needed.
	Anemia, poor nutrition, other organ failure, uremia, insomnia	
Itch 84%	Dry skin	Emollients for dry skin.
	Secondary hyperparathyroidism Hyperphosphatemia Anemia Opioids	Antihistamine such as diphenhydramine (can worsen fatigue and delirium). Steroid.

Physical symptom	Cause	Treatment (see CHAPTER 2 for details)
Dyspnea (the most distressing symptom) 80%	Pulmonary edema Pleural effusion Metabolic acidosis Anemia Aspiration Comorbid CHF, COPD, or other lung pathology	If comfort and quality of life are the only goals of care, not preservation of renal function, diurese for symptomatic pulmonary or peripheral edema. Opioid relieves dyspnea of any cause. Renal dosing for morphine (see **APPENDIX A**).
Delirium (agitated or hypoactive) 76%	Metabolic derangements Hypoxia/hypercapnia Medications	Reduce or eliminate culprit medications, if possible. Haloperidol.
Pain 73%	Bone pain due to 2° hyperparathyroidism Diabetic neuropathy Polycystic kidney disease Calciphylaxis (hemodialysis patients only)	Avoid NSAIDS due to nephrotoxicity and increased risk of bleeding with uremic platelet dysfunction. Paracetamol is safe and requires no dose adjustment for renal failure. Morphine: active metabolite accumulates in CKD 5 and can cause neurotoxicity. Use lower dose and/or longer dosing interval than usual.
Anorexia 71%	Nausea (see below) Diabetic gastroparesis Dry mouth	Anti-emetics (see below). Metoclopramide for gastroparesis. Oral care. Steroid to stimulate appetite.
Swelling in legs/arms 71%	Fluid overload Low oncotic pressure due to proteinuria and malnutrition Comorbid heart failure	Elevate edematous extremities (although this may exacerbate pulmonary edema). Elastic wraps as tolerated. If comfort and quality of life are the only goals of care, not preservation of renal function, diurese for symptomatic peripheral edema or anasarca. This may require higher than normal furosemide doses.
Dry mouth 69%	Intravascular volume depletion (can exist even with total body fluid overload).	Instruct family caregivers to keep mouth moist with sips of liquid or sponge.
Constipation 65%	Medications including opioids and anticholinergics Intravascular volume depletion	Laxative
Nausea with or without vomiting 59%	Uremia Emetogenic medications Diabetic gastroparesis	Reduce or eliminate culprit medications, if possible. Haloperidol for nausea due to endogenous or exogenous emetogenic toxin. Start with low dose. Metoclopramide for gastroparesis.
Cough 47%	Pulmonary edema Aspiration Comorbid COPD or other lung pathology	If comfort and quality of life are the only goals of care, not preservation of renal function, diurese for symptomatic pulmonary edema. Opioid relieves cough of any cause. Renal dosing for morphine.

TABLE 6.7 Common Psychological Symptoms in Advanced Chronic Kidney Disease and Their Treatment

Psychological symptom	Treatment
Anxiety 78%	Psychosocial supports Haloperidol Diazepam (can cause delirium and paradoxical agitation)
Feeling sad 65%	Psychosocial supports
Depression Unknown prevalence	Psychosocial supports Antidepressant such as fluoxetine or amitriptyline

Many medications, including those used for palliation, require dose adjustment for patients with renal failure (see **APPENDIX A**).

6.7 Renal Replacement Therapy (Hemodialysis and Peritoneal Dialysis)

Without renal replacement therapy, patients who have end-stage renal disease (CKD 5) will die in a matter of days or weeks. Death results from electrolyte imbalances such as hyperkalemia, which causes fatal cardiac arrhythmias or suffocation from fluid overload. These deaths can be witnessed in district hospitals across sub-Saharan Africa. In the days before death, patients may also suffer intractable nausea and vomiting, mental status changes, and seizures as a result of the buildup of urea and other toxins.

Dialysis offers an effective and immediate solution to otherwise imminent death from renal failure. Dialysis may be performed by filtering the blood through a membrane (hemodialysis), or by filtering the blood across the peritoneum (peritoneal dialysis).

Unfortunately, hemodialysis is technically demanding and the consumable costs of both hemodialysis and peritoneal dialysis remain high. Peritoneal dialysis is a preferable option in resource-limited settings. Hemodialysis can only be offered at referral centers because of the technical demands of the procedure (including water filtration and equipment maintenance). Peritoneal dialysis can—in theory—be delivered at local facilities or in the patient's home. The risk of peritoneal infection in peritoneal dialysis equals the risk of venous catheter infection in hemodialysis.

Dialysis of either kind is currently performed in Rwanda only at referral centers. The primary indication is for acute, reversible renal failure. We do not currently have an estimate of the number of patients in Rwanda who would require chronic dialysis, if available.

Barriers to more extensive availability of chronic peritoneal dialysis include the costs of the solution. Centers in India have been able to provide even hemodialysis for $2500 per year for twice-weekly sessions. Continuous ambulatory peritoneal dialysis costs $4500 per year for 3 exchanges per day.[19] These examples give hope that this service can be provided at an affordable price, using less expensive consumables.[20,21]

6.8 Renal Transplantation

Renal transplant has been shown to represent a better value than dialysis, in terms of money spent and quality of life gained. Middle-income countries such as India and South Africa provide one avenue for patients in lower-income countries to undergo life-saving renal transplant surgery. This may be possible only if patients have living donors who can travel with them. The high cost of immunosuppressive drugs poses another barrier to successful kidney transplantation. Older immunosuppressives such as cyclosporine are now generic, but annual costs remain around $2000 per year for typical regimens. Other issues still to be resolved are appropriate systems of follow-up for transplanted patients in resource-limited settings.

In our opinion, any transplant program should be conducted nationally to ensure a fair and equitable process of case selection. This is the same strategy used for cardiac surgery patient selection in Rwanda. Given the limited number of transplants needed in a country like Rwanda, the annual cost per capita of such a service may not be prohibitive, if organized efficiently.

6.9 Acute Kidney Failure in Hospitalized Patients

This manual does not address management of acute renal failure in hospitalized patients. We refer to the forthcoming WHO district clinician manual for adolescents and adults.

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