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Emergencies in children with chronic renal failure (CRF), although uncommon, represent a special clinical entity that requires rapid recognition and appropriate management by the emergency physician.

The challenge with these children is to identify both reversible causes of chronic renal failure which— if left untreated—will further compromise their renal function, and potentially life-threatening conditions that require immediate interventions.

Current practices and recommendations regarding management have changed significantly during the past few years. For example, intravenous or inhaled albuterol has been shown to be an effective, rapid, and safe treatment for moderate hyperkalemia in children.

Appropriate management of these emergencies requires a knowledge of the basic pathophysiology and treatment regimens currently being used for children with CRF, since many of these emergencies may be treatment-related.

Emergency department (ED) physicians must be familiar with advances in dialysis techniques and indications as well as complications that may be associated with the use of this technology. In addition, the increasing use and success of renal transplantation has created a subgroup of pediatric patients with special needs that must be identified and met in the ED.

Each child with chronic renal failure presenting to the ED should be carefully evaluated and final disposition made in conjunction with the nephrologist.

— The Editor

Emergencies in Children with Chronic Renal Failure

Author: **Georges Ramalanjaona, MD, DSc, FACEP**, Associate Professor of Emergency Medicine, Associate Chairman for Academic Affairs, Department of Emergency Medicine, Seton Hall University School of Graduate Medical Education, South Orange, NJ.

Peer Reviewer: **Stephen R. Knazik, DO, FACEP**, Assistant Professor, Departments of Pediatric and Emergency Medicine, Wayne State University School of Medicine, Detroit, MI.

Introduction

Chronic renal failure (CRF) is defined as an irreversible and progressive reduction in the glomerulofiltration rate (GFR) to below 25% of normal level (decline of 30 mL/min/1.73 m²) for at least three months.[1] Creatinine clearance (CCr) is a good indicator of GFR and is helpful in monitoring renal function of children in various age groups. *(See Table 1.)*

The estimated incidence of CRF ranges from one to three children per million in a population younger than 16 years of age. Complications of CRF vary with the degree of renal insufficiency and the nature of primary renal disease. In children younger than 5 years, congenital renal diseases, such as renal hypoplasia, renal dysplasia, and obstructive uropathy, are the most common cause of CRF.[2] In older children, hereditary diseases, metabolic diseases and acquired etiologies occur more frequently. Hereditary diseases include juvenile nephritis, cystic kidney, and Alport syndrome. The most frequent metabolic causes are cystinosis and oxalosis, and

the principal acquired etiology is chronic glomerulonephritis.[3,4]

Pathophysiology

Emergencies in CRF constitute a special entity in the pediatric population. To understand its development, the emergency physician (EP) must understand the basic pathophysiology leading to this disease.

The kidney plays an essential role in maintaining homeostasis. Thus, renal dysfunction affects electrolytes and water balance, acid-base balance, blood pressure control, calcium and phosphate metabolism, hemoglobin level, and the clearance of endogenous toxins, known as azotemia. The classic signs of CRF occur as each of these systems is affected. *(See Table 2.)*

The precise mechanisms resulting in progressive, irreversible functional deterioration of the nephron remains unclear; however, several factors may play critical roles, including immunologic injury, dietary protein and phosphorus intake, hemodynamically mediated hyper-filtration in the remaining glomeruli, and systemic hypertension with persisting proteinuria.[5]

Table 1. Mean CCr Values

NORMAL VALUES

Newborn: 38 mL/min/1.73 m²

At 1 year of age: 77 mL/min/1.73 m²

Between 4 and 10 years: 109 for female, 124 for male

Adult values (for reference): 117 for female, 131 for male.

Deposition of immune complex or anti-glomerular basement membrane antibodies in the glomerulus may result in persistent glomerular inflammation leading to sclerosis.

Experimental and human studies of chronic renal insufficiency have shown that a high-protein diet accelerates the development of renal failure.[6] Conversely, a diet low in protein and phosphorus reduces the functional deterioration of nephrons. Although the exact mechanism is still unclear, once renal function starts to decline, compensatory mechanisms develop in the remaining nephrons to maintain a normal homeostasis (internal environment). However, when the GFR drops below 25% of normal values, complex clinical, biochemical, and metabolic abnormalities occur that constitute the uremic state.

Hyperfiltration injury in the surviving glomeruli may represent an important, final, common pathway of glomerular destruction, regardless of the initial mechanism of renal injury. When nephrons are lost, the surviving ones undergo a structural and functional hypertrophy mediated by an increase in glomerular blood flow. This high hydrostatic pressure is associated with dilatation of afferent arterioles and vasoconstriction of efferent arterioles and will result in changes in the integrity of capillary wall, leading to an increase in passage of proteins across the wall. This will result in changes in mesangium and epithelial cells, with the development of glomerular sclerosis. As sclerosis progresses, the surviving nephrons show an increase in a number of fuctions. Their excretory functions result in a vicious cycle of increasing glomerular blood flow and hyperfiltration.

Finally, systemic hypertension from any etiology or persistent proteinuria directly may affect the glomerular capillary wall, leading to sclerosis and more hyperfiteration injury.

Dialysis

Dialysis remains one of the standard treatments of CRF. However, when it is delayed or improperly applied, complications may ensue; thus, the EP should know the different modalities of dialysis in children and its common indications and complications.

A GFR of less than 5% of normal is usually an indication for dialysis treatment in children with CRF. It is used to regulate solutes and fluid abnormalities. Common indications for emergent dialysis in this subset of the population include fluid overload (pulmonary edema/congestive heart failure [CHF]), hyperkalemia, severe hyponatremia, metabolic acidosis, malignant hypertension, bleeding diathesis, and uremic encephalopathy. Less frequent indications are drug overdose, severe hypocalcemia or hypermagnesemia, cardiac tamponade, and pericarditis.

There are two major dialysis modalities: peritoneal dialysis (PD) and hemodialysis. Each is based on the principle of filtering the patient's blood through a semi-permeable membrane bathed

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Editorial Group Head: Valerie Loner
Managing Editor: Suzanne Zunic
Marketing Manager: Schandale Kornegay

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Table 2. Classic Manifestations of CRF

- Azotemia
- Metabolic acidosis
- Anemia
- Bleeding diathesis
- Infection
- CNS changes
  - Altered mental status
  - Peripheral neuropathy
- Hypertension
- Pericarditis
- Hyperkalemia
- Growth retardation
- Renal osteodystrophy
- Sodium retention/wasting

in a balanced physiologic solution. Because of osmotic gradients between these two fluids, water and solutes will diffuse across the membrane, thus normalizing the patient's blood composition.

Peritoneal Dialysis. PD requires the insertion of a catheter into the peritoneal cavity for the instillation of a dialysis solution for 4-8 hours, 4-5 times a day. The retained body solute will diffuse from the blood to dialysate via the peritoneum.

Two types of PD are available for children with CRF: continuous ambulatory peritoneal dialysis (CAPD) and continuous cyclic peritoneal dialysis (CCPD).[7-10]

CAPD is the standard technique for the majority of the pediatric population. It is a continuous procedure that has the advantage of allowing the maintenance of satisfactory levels of blood urea nitrogen (BUN) and creatinine, smoother control of fluid and hypertension, and avoidance of the use of anti-coagulants.

In CAPD, dialysis across the peritoneal membrane removes excess body water through an osmotic gradient created by the glucose concentration in the dialysate. Resulting wastes are removed by diffusion from the capillaries into the dialysate. The peritoneal cavity is accessed by the insertion of a Tenckoff catheter via a midline infra-umbilical incision. Then, the catheter is brought out through the skin by a subcutaneous tunnel and connected to an extension tube with a spike for insertion into the dialysis bag. Family members are taught this technique of spiking the bags of dialysate, allowing the dialysate to run and remain in the peritoneal cavity for the allotted period, and then draining the dialysate into the bag. Such exchanges are performed on a regular basis (usually 3-5 times a day) over a 24-hour period. Since the benefits of CAPD far outweigh the risks (see Table 3), it still remains the optimal form of chronic dialysis for the majority of children.

CCPD is an acceptable and equally effective alternative to CAPD, and it uses the same principle as CAPD. However, this procedure reverses the schedule of CAPD by allowing automatic exchanges at night only, using a simple cycler machine. The use of this device permits an uninterrupted day of activities, a decrease in the number of connections and disconnections, less time spent to perform dialysis, and a reduction in parental burnout.[11]

Hemodialysis (HD). In this modality, the patient's heparinized blood is pumped through an extra-corporal circuit where it comes into contact with an artificial membrane across which fluid and solute movement occurs. The amount of fluid transferred can be controlled by adjusting the pressure under which blood is pumped through the dialyzer. This technique requires special vascular access to the patient's circulation through a surgically created arterio-venous fistula or an implanted artificial graft. This vascular access should be treated with caution, since careless manipulation can result in bleeding, infection, or thrombosis and lead to a loss of the access. HD is usually performed three times a week for 3-5 hours per session, either at home or at a specially staffed dialysis unit.[12,13]

Emergencies in Children with CRF

In the ED, clinical manifestations of CRF in children present serious challenges for the EP's diagnostic skills due to a variety of reasons, including:

1. A child with a new diagnosis of CRF, especially one who has not yet started dialysis, often presents to the ED with non-specific complaints of insidious onset (i.e., weakness, poor appetite, and altered mental status), thus masking the etiology and severity of the condition.

2. Although CRF is typically irreversible and progressive, the EP must exclude potentially reversible factors and identify treatable causes of CRF (see Table 4), which if properly managed, may allow for some return of renal function. Furthermore, with an early diagnosis and aggressive management, the EP may be in the unique position of making a significant difference in the patient's outcome.

3. Also, in the context of a pediatric patient with CRF presenting to the ED with an acute problem, the EP should promptly identify and manage intercurrent illness that results in clinical decompensation of CRF, and restore the patient to a stable compensated state. Therefore, these conditions need to be treated vigorously and in a timely manner. These reversible factors include:

- Volume depletion, which will lead to decreased renal perfusion in the already impaired kidney's ability to concentrate fluid and solute.
- Increased protein catabolism secondary to stressful events such as intercurrent infection, trauma, surgery, or gastrointestinal bleeding, which are responsible for worsening azotemia.
- Cardiac insufficiency of any etiology may decrease renal perfusion and further deteriorate renal function.
- Accelerated hypertension (HTN) with severe vasoconstriction may rapidly impair renal function, as in patients with scleroderma, that can be readily reversed with angiotension-converting enzyme (ACE) inhibiting drugs.

Side effects of the drugs themselves constitute an important group of reversible factors.

Diuretics may exacerbate renal insufficiency by causing volume depletion. Anti-hypertensive agents (i.e., ACE inhibitors) may decrease renal function in patients with bilateral renal stenosis. Non-steroidal anti-inflammatory drugs (NSAIDs) may produce acute interstitial nephritis. Radiocontrast agents can cause acute tubular necrosis.

Post-renal reversible causes are important because of their

Table 3. Benefits and Risks of CAPD

BENEFITS	RISKS
• Technical simplicity	• Catheter malfunction
• Cheaper than hemodialysis	• Infection
• Fewer blood transfusions than hemodialysis	• Parental burnout
• Minimal dietary restrictions	• Hyperlipidemia
• Better/steady control of solutes	
• Greater flexibility	

Table 4. Treatable Causes of CRF

- Malignant hypertension
- Renal artery stenosis
- Acute interstitial nephritis
- Hypercalcemic nephropathy
- Multiple myeloma
- Vasculitis
  - Lupus erythematosis
  - Polyarteritis

frequency. They include reflux nephropathy and papillary necrosis in children with diabetes mellitus, sickle cell disease, or a history of long-term use of analgesics.

4. Finally, clinical presentations of CRF in children may manifest as potentially life-threatening conditions even in the dialyzed patient. They include: volume overload; metabolic disturbances (i.e., hyperkalemia, hyponatremia, and metabolic acidosis); hypertensive crisis; infection; complications related to treatment; peritonitis; PD catheter malfunction; graft emergencies; post-dialysis problems (i.e., hypotension); and fever and rejection in post-transplant patients.

The following sections review the clinical manifestation of these true emergencies and outline their ED management.

Volume Overload

The most common emergency complaint in children with CRF is fluid overload, and it may manifest with a variety of complaints, including pulmonary edema or CHF. Frequently, the etiology is extra fluid or salt in excess of the patient's greatly diminished excretory capacity. A decrease in the kidney's filtering capacity usually does not result in volume overload unless the GFR is less than 10% of normal for age.[14]

Typically, the child presents to the ED with dyspnea on exertion or paroxysmal nocturnal dyspnea. Further history reveals recent weight gain (more than 5 lbs) and weakness, and physical examination may show the expected signs of pulmonary edema or CHF, which may be confirmed by chest radiograph.

However, presenting signs may be subtle (except for weight gain and increasing dyspnea), and in the absence of other etiology of dyspnea in CRF, the EP should assume the diagnosis of volume overload and treat the patient accordingly.

Temporizing measures which may be instituted include:

- Placement of the child in a sitting position, with administration of high flow oxygen and fluid restriction;
- IV or sublingual nitroglycerin (NTG) to decrease both preload and afterload;
- IV nitroprusside as an alternative to NTG. It may be more effective in producing arteriolar dilatation than NTG; and
- IV morphine to decrease preload. Its routine use as a first-line drug in pulmonary edema has become less frequent;
- IV diuretics (i.e., furosemide at a dose of 1 mg/kg/dose). Although less effective in patients with advanced CRF, it is still a powerful pulmonary venodilator, and its use may result in relief of dyspnea. In addition, patients with residual renal function may respond to a large IV dose of furosemide.

The approach to treatment of volume overload in patients with CRF is different from that for children with normal kidney function, in that the main priority is to arrange for an emergent dialysis, which is currently the most effective means of reducing intravascular load in the absence of renal function.

In summary, temporary measures may be utilized, in consultation with the child's nephrologist, to treat volume overload while awaiting dialysis or to delay dialysis in a patient with residual renal function. The EP and the pediatric nephrologist should discuss definite treatment and appropriate disposition.

Metabolic Disturbances

Major metabolic crisises that may be encountered include hyperkalemia, hyponatremia, and metabolic acidosis.

Hyperkalemia. This is potentially the most rapidly lethal complication of CRF, especially since it is usually clinically silent until it presents with life-threatening manifestations. Therefore, the EP must look for hyperkalemia in any child with CRF, especially when GFR falls below 5% of normal for age.

Hyperkalemia can be exacerbated even by modest exogenous and endogenous loads. The use of drugs such as ACE inhibitors and beta-adrenergic blockers, which usually have only minimal effects on serum K^+ in normal subjects, may have devastating consequences in a child with deteriorating renal function.

When hyperkalemia is suspected, an electrocardiogram (ECG) should be rapidly obtained to look for electrical signs of hyperkalemia (peaked T waves, widened QRS complexes, flattened P waves, ectopic rhythms, and intra-ventricular block). If electrical changes are present, then appropriate treatment should be initiated before laboratory confirmation of hyperkalemia. Also, ECG changes may be absent when hyperkalemia is severe; therefore, a normal ECG does not make laboratory confirmation of serum K^+ level unnecessary.[15,16]

A serum K^+ greater than 6 mEq/L should be evaluated for its accuracy and signs of cardiac toxicity, since false increases of 1-2 mEq/L in serum K^+ may be due to hemolysis of the specimen or to local muscle release of K^+ from prolonged tourniquet application. However, when in doubt, it is appropriate to treat hyperkalemia while awaiting the results of a repeat potassium test since reducing a normal potassium level rarely causes any adverse events, whereas delaying treatment for hyperkalemia can be potentially harmful, even fatal. In the case of a patient with known CRF who presents to the ED in cardiac arrest, hyperkalemia should be assumed and treated accordingly.

The most rapidly effective treatment is IV administration of 10% calcium gluconate 0.2-0.5 mL/kg slowly over 2-5 minutes for the presence of electrical signs of hyperkalemia. Calcium

transiently reverses the ECG signs of hyperkalemia without changing the serum K^+ level or total body K^+.

The next temporizing measure consists of administering IV sodium bicarbonate 1-2 mEq/kg and/or IV glucose D50W 0.5-1 gm/kg followed by infusion of D25W with IV insulin 1 unit per 4 gm of glucose infused to maintain blood sugar between 120 and 300 mg/dL. Use D10W in a child weighing less than 10 kg, and D25W in a child weighing 10-30 kg. Intravenous glucose and insulin drive potassium into cells, thus reducing the serum potassium level. This combination is effective, but requires more time to achieve the desired effects than intravenous sodium bicarbonate. However, repeated doses of bicarbonate should be avoided, since it may precipitate fluid overload in a CRF patient with limited ability to tolerate volume and solute.[17]

The use of inhaled or intravenous albuterol recently has been advocated to shift potassium into cells and lower the serum potassium by 1-1.5 mEq/L. The authors of several clinical trials concluded that this drug administered via nebulizer or intravenously is an effective, rapid, and safe treatment for hyperkalemia in children and advocated its use as a first-line emergency treatment for this disorder in their centers.[18-22]

All of these temporary measures are helpful in altering the distribution of potassium or decreasing the cardiac sensitivity to hyperkalemia, but they do not diminish the total body's potassium content. The total body potassium can be decreased by administration of the exchange resin sodium polystyrene sulfonate (Kayexlate), which is a resin that exchanges sodium for potassium in colonic mucosa. Each gram of resin/kg of body weight will decrease the serum potassium level by 1 mEq/L. Kayexlate may be administered orally or rectally. It is usually given with sorbitol (20% solution) to decrease the associated constipation, which affects its efficacy. The rectal administration acts more rapidly to decrease potassium levels, and should be given as an enema to ensure prolonged contact between the colonic mucosa and drug. Since administration of this medication results in potassium being exchanged for sodium, the process may result in a significant sodium burden, which may lead to hypertension (HTN) or volume overload in CRF children.

While these temporizing maneuvers are necessary, more definite measures to decrease potassium load, such as dialysis, should be discussed with the pediatric nephrologist.

Hyponatremia. Hyponatremia may cause malaise, altered mental status, or seizures in a child with CRF. It is almost never due to sodium depletion, but rather purely dilutional since total body sodium is usually normal. Treatment consists of water restriction, hypertonic saline, and urgent dialysis.[23,24]

Metabolic Acidosis. Acidosis will frequently occur in children with CRF and should not be corrected unless the serum bicarbonate falls below 20 mEq/L.[25]

Treatment consists of intravenous infusion of sodium bicarbonate to achieve a pH greater than 7.2 and a serum bicarbonate level above 20 mEq/L. The amount of bicarbonate required may be calculated by this formula:

Base deficit = 0.6 × body weight in kg × (mEq/L desired HCO_3 Level - observed level), divided by 2.

The EP should give half of the replacement in the first 2-3 hours and then infuse the remaining amount over 22 hours. As previously noted, children with CRF may not tolerate the administration of this quantity of sodium. For these situations an acceptable alternative is the use of thromethamine (Tham) in 0.3 m solution.

The correction of significant metabolic acidosis will necessitate the admission of the child to the appropriate inpatient unit for close monitoring.

Hypertensive Crisis

The prevalence of HTN in childhood is between 0.5% and 5%, and renal disease is the most common cause.[26] Although the true definition of HTN in the pediatric population remains controversial, the accepted definition of blood pressure (BP) norms comes from the 1987 report on the second task force on BP control in children from the National Heart, Lung, and Blood Institute.[27] The resulting figures, correlated with height-weight, are based on experiences, and consensus and may be used in the ED.

Definition. Normal blood pressure is defined as a systolic and diastolic less than the 90th percentile for age- and sex-specific norms, high normal as between 90th and 95th percentile, and HTN as systolic and diastolic equal and greater than 95th percentile. The task force further defines two classes of HTN in children: Significant hypertension is between the 95th and 99th percentile, and severe hypertension is at or above the 99th percentile. In order to establish HTN, measurements should be obtained on at least three readings, and the correct BP cuff size must be used.

Physiology and Etiology. The physiologic basis of HTN depends on the underlying cause, and it is estimated that in 79-98% of cases, pediatric HTN is usually secondary to a specific etiology, the most frequent being renal (80%).

Renal HTN is caused by vasoconstriction to the afferent arteriole of the glomerulus, resulting in the secretion of renin from the juxtaglomerular complex. This leads to aldosterone secretion, which results in sodium retention and potassium loss in the kidneys. Sodium retention produces hypervolemia and increased cardiac output, which leads the renin-angiotensin system to increase peripheral vascular resistance, which increases the HTN.

HTN may be the presenting sign or complication of significant renal diseases, including chronic infection, post-streptococcal glomerulonephritis, reflux nephropathy, hemolytic-uremic syndrome, and anaphylactic purpura.

Following renal disease, the second leading cause of secondary HTN in children is coarctation of aorta (2%). Even after surgical correction, HTN may persist secondary to the hyperdynamic state, persistent pressure gradient, and increased sympathic tone, based on how long the condition went unimpaired.

Endocrine causes (i.e., pheochromocytoma) represent an important but a small percentage of children with HTN.

HTN can also be secondary to acute stress situations such as major trauma, burns, central nervous system (CNS) infections, and autonomic dysfunction.

Drug ingestion should always be considered in any unexplained HTN in children.

Essential or idiopathic HTN is also a possibility in the pediatric population, and is probably the result of interaction of genetic, environmental, and constitutional factors.

Treatment. The decision to treat HTN in children with CRF is based on a combination of BP readings and symptomatology.[14,28,29]

In a child with chronic HTN, higher blood pressures may be necessary to maintain end-organ perfusion. Thus, a rapid decrease in BP may compromise perfusion of vital organs (i.e., brain, heart, and kidneys). The goal of treatment is to limit cardiovascular sequelae as well as diminish the rate of progression of CRF with a reduction of BP to the 50th percentile for age or 15-25% in mean arterial pressure.

In severe HTN, appropriate treatment is indicated to prevent hypertensive encephalopathy, stroke, or CHF. Since HTN in children with CRF is due to expanded vascular volume because of salt and water retention, and activation of renin-angiotensin system, any treatment of HTN in those children should include salt and water restriction and/or diuretics, and other anti-hypertensive drugs targeted at these two mechanisms.

Most children will require anti-hypertensive drugs as a single agent or in combination with others to effectively control HTN in the ED. As a rule of thumb, a single agent should be initiated at less than maximal dose, then increased to its maximum before adding a second drug. Careful and regular monitoring of BP is necessary to avoid rapid decreases in BP that may further compromise the GFR. A wide variety of agents can be used in the ED to effectively control HTN.

ACE Inhibitor Agents. This class of agents is useful for the treatment of renin-mediated HTN, which is frequent in children with CRF. Captopril is particularly useful in children who develop hypertension following the use of an umbilical artery catheter. Disadvantages of this medication include slow incremental dosing, which limits its use in the ED, and its potential to precipitate acute renal failure (ARF) in patients with renal arterial stenosis. The initial dose is 0.3 mg/kg orally, doubled every two hours to a maximum of 2-6 mg/kg or until the desired effect is achieved.

Enalapril is an acceptable alternative, since it has fewer side-effects and its dose is one-tenth that of captopril. However, serum potassium should be monitored with administration of this medication because it may result in hyperkalemia.

Diuretics. A trial of diuretics to stimulate remaining renal function may be useful in the hypertensive child with volume overload. Furosemide can be used at a dose of 1 mg/kg every two hours with the maximum effect usually within 30 minutes.

Beta-Adrenergic Blocking Agents. The most frequently used drug of this class is labetalol, a combined alpha- and beta-blocker, that is effective for the treatment of significant/severe HTN. An initial loading dose of 0.2-1 mg/kg is administered intravenously and followed by escalating doses of 0.5-1 mg/kg, followed by a constant infusion of 1-3 mg/kg/hour. It is contraindicated in patients with bradycardia, bronchospasm, or CHF.

Calcium Channel Blockers. In general, as these agents decrease BP, vascular volume increases, thus requiring the use of diuretics. Furthermore, vasodilatation results in tachycardia, which necessitates the concommitant use of beta-blockers.

The choice of an agent depends on severity of HTN and size of the child.

The short-acting nifedipine acts within 30 minutes when given orally or rectally, but results in wide variation of BP. The dose is the same as the long-acting preparation.

Other Agents. Minoxidil is a very potent agent, usually effective in children with refractory HTN, at dose of 0.1-2 mg/kg. The most significant side-effects are hypotension and hirsutism.

Intravenous nitroprusside is both an arterial and venous dilator and is reserved for severe or refractory HTN. Patients usually require continuous BP monitoring via an arterial line, and admission to an ICU. The drug is given at a continuous infusion rate of 0.1-10 mcg/kg/min, and BP should be titrated against infusion rate as the onset of action is within a few seconds. If hypotension occurs, the infusion should be slowed or stopped. Cyanide toxicity is one side effect that is increased in CRF, and can cause metabolic acidosis.

Infection

Infection is a leading cause of morbidity and mortality in children with CRF. These children have an increased susceptibility to infection most likely secondary to defects in both cellular and humoral immunity. Thus, the potential for serious infection should always be entertained even when the classic signs are not evident. For instance, the presence of fever alone may be due to occult bacteremia or sepsis, and pneumonia may present as atypical dyspnea, which must be differentiated from volume overload. In the setting of a child with fever or dyspnea, all diagnoses should be considered, ancillary tests performed, and children placed on empiric broad-spectrum antibiotics (dose corrected for CRF) until the proper etiology has been established.

Emergencies Related to Treatment

Peritonitis. Peritonitis remains the primary complication of long-term peritoneal dialysis in children. The incidence of peritonitis in CCPD is one episode per year, with a slightly higher rate in CAPD.

The potential routes for peritoneal contamination include the lumen of the peritoneal catheter, the sinus tract around the catheter, the blood stream or peritoneal lymphatics, and across the walls of an intraperitoneal hollow viscus.[30] Important factors that influence the development of peritonitis include skin infections of the hand, infection of sites in proximity to the catheter exit site, or catheter exit site infection.

The signs and symptoms of peritonitis include fever, vomiting, diarrhea, and abdominal pain or tenderness. The peritoneal fluid is usually cloudy. A peritoneal fluid sample is obtained prior to initiation of antibiotics, and sent for Gram's stain, cell count, and cell culture. A WBC of more than 100 cells/cm with more than 50% neutrophils is suggestive of peritonitis.

The majority of infections (60-70%) are caused by gram-positive cocci (*Staphylococcus epidermidis and Streptococcus viridans*) which frequently occur at the exit site. The most serious form of peritonitis is fungal peritonitis and is commonly associated with an episode of bacterial peritonitis and use of antibiotic therapy in the preceding month.[31,32] Microscopic examination of peritoneal fluid using gram stain-identified yeast infections with budding yeast or pseudohyphae in up to 30% of cases. Fungal peritonitis is significantly associated with morbidity and mortality, and commonly results in abdominal adhesions that ultimately destroy the functional ability of the peritoneal membrane to dialyze.[33]

In a patient with CRF and abdominal pain it is important to remember that peritoneal dialysis patients are still at risk for serious intra-abdominal events such as appendicitis, pancreatitis, and cholecystitis, which are especially important to consider when the peritoneal culture reveals multiple organisms.

The goals in treatment of peritonitis include elimination of peritoneal infection, preserving the integrity of the peritoneal membrane, and not aggravating the child's underlying condition.

The accepted therapeutic regimen consists of peritoneal lavage and antibiotic administration. When peritonitis is identified, the care giver should perform three rapid flushes with peritoneal dialysate and heparin (500 U/L.)

Most peritonitis episodes can be treated with intra-peritoneal antibiotics (IP) or with intravenous antibiotics in moderate to severe cases. Antibiotics which are useful in the treatment of peritonitis include penicillin G, gentamycin, vancomycin, clindamycin, and tobromycin. Practically, the choice of an initial antibiotic is guided by the gram stains of the peritoneal fluid, which are positive in 30-50% of cases. Antibiotic therapy is then modified according to the findings of the peritoneal dialysis fluid culture. Most antibiotics (with the exception of Penicillin G) retain at least 75% of their bioactivity when used in dialysate solute.

When patients develop antibiotic resistant peritonitis, or fungal peritonitis, hospitalization is indicated. The Tenckoff catheter should be removed (even if functioning), surgical consult obtained, and hemodialysis initiated.

Peritoneal Dialysis Catheter Malfunction. These technical difficulties are commonly encountered and may be simple or complex. The two most frequent complications are leakage and obstruction.

Leakage. The incidence of leakage varies from 5% to 25%. Risk factors include previous abdominal surgery (i.e., previous catheter insertion) debilitated or obese patients, and children treated with corticosteroids.

Early leakage leads to infection at the exit site and in the subcutaneous tissue. Late leakage may result in peritoneal tears that lead to fluid dissecting into the abdominal wall, scrotum or vagina, or chest wall.

Minimal leakage usually does not necessitate catheter removal as it usually spontaneously resolves. Late or significant leakages may require discontinuation of dialysis to allow the peritoneal tear to heal (about 8-10 days). During this time patients will benefit from short-term hemodialysis. Patients who develop a hydrothorax (incidence 2%) are managed by cessation of the peritoneal dialysis and temporary use of hemodialysis. In some instances, thoracotomy may be required to repair the defect.

Overflow Obstruction. One of the main mechanical complications of peritoneal dialysis is failure of the dialysate to drain. Major causes include catheter migration and fibrin clots. Catheter migration usually leads to removal of the catheter in 90% of cases. Fibrin clots may occur as a result of bleeding after catheter implantation or after an episode of peritonitis.[34,35]

Large amounts of heparin (i.e., 5000 U) or thrombolytic drugs in a small volume of dialysis can be used to dissolve the fibrin clots during an irrigation of the catheter. If successful, subsequent exchanges are done with 1000 units of heparin added to each liter of dialysate. Catheter replacement is necessary when the above attempts have failed.

Graft Emergencies

Graft emergencies constitute special emergencies necessitating both technical knowledge of hemodialysis and timely diagnosis and management of its potential complications.

Hemodialysis requires the ability to access the vascular space and to provide significant extracorporeal blood flow. The most commonly used access to the pediatric patient weighing more than 20 kg is an arterio-venous (AV) fistula. There are two types of AV fistulas which are used: an autogeneous fistula or a synthetic graft. Autogeneous fistulas surgically anastomose the cephalic vein and radial artery using a side-to-side vein to artery connection in the non-dominant arm, called a brachio-cephalic fistula. A synthetic graft can also be utilized in the arm with either a straight or loop configuration. These synthetic grafts include polytetrafluroethylenl, PTFE Gore-Tex, or Impra. In general, autogenous fistulas last longer than synthetic grafts and are less prone to thrombosis, stenosis, and infection. Young children and infants have smaller size vessels for AV fistulas and require vascular access in the subclavian or internal jugular vein for hemodialysis.[36-38]

Common emergencies that occur with hemodialysis include bleeding, thrombosis, and infection. The occurrence of these complications mandate vascular surgery and pediatric nephrology consultations.

Persistent Bleeding. Surgical trauma or technical errors may result in persistent bleeding in 30% of patients. In most cases, the bleeding spontaneously subsides. Instances that fail to resolve may require transfusion and surgical repair.

The necessity of infusing anticoagulants (i.e., heparin) in hemodialysis patients predisposed to bleeding may also lead to persistent bleeding. Clinical manifestations include petechial skin hemorrhage, sub-capsular liver hematoma, retroperitoneal hematoma, and gastro-intestinal bleeding. Treatment consists of supportive therapy, transfusion, and altering the patient's heparinization prescription (i.e., low dose, intermittent, or regional heparin) or starting on peritoneal dialysis. Studies have reported the successful use of hemodialysis without anticoagulant during the perioperative period in adults, but data are not yet available for the pediatric population.[37]

External blood loss may be due to breaks in the dialyzing membrane, manufacturing defects, or separation of blood lines. Most membrane leaks are due to rupture of a dialysis membrane and require clamping of arterial and venous blood connections and discontinuation of dialysis. The EP's decision to transfuse blood will depend on the amount of blood loss, clinical severity of symptoms, and the pre-dialysis hemoglobin level.

When separation of the blood line is a cause of blood loss, reconnection and continuation of dialysis treatment is feasible.

Thrombosis. Thrombosis is a common cause of loss of vascular access and should be rapidly recognized with every effort made to salvage the access. Many factors have been implicated in the genesis of thrombosis.

Hypercoagulable states (i.e., clotting factor abnormalities) are similar to those seen in children with nephrotic syndrome, including low serum protein, high levels of fibrinogen and factor VII, and reduced half life for fibrinogen may exist. If the clotting defect is diagnosed prior to dialysis, correction of the abnormal factor may be achieved prior to initiation of dialysis.

Technical factors during surgical anastomosis may result in thrombosis and should be corrected as soon as possible to prevent loss of the vascular access. The diagnosis is established clinically by the loss of the thrill or bruit over the anastomosis. Surgical repair is the usual treatment.

Insufficient or inadequate heparinization prior to or during

dialysis may result in thrombosis. This condition is easily corrected through adjusting to the optimal dose during each dialysate.

Late-graft thrombosis (after 48 hours) is usually caused by anastomotic failure or infection. Late anastomotic failure is produced by intimal hyperplasia on the distal graft which in turn facilitates the formation of clots. Treatment consists of intravenous thrombolytic therapy to dissolve the clots or surgical thrombectomy.

Graft Infection. Graft infection will result in a non-functional anastomosis with thrombosis, which is commonly managed by intravenous antibiotics and graft removal.

Bacterial contamination, usually gram-positive cocci, may occur during surgery. A culture and sensitivity from the site should be obtained, and the patient should receive intravenous antibiotics. Consultation with the surgeon who operated on the patient should be obtained.

Post-Dialysis Problems

Hypotension, muscle cramps, and dialysis dysequilibirum are all potential post-dialysis complications.

Hypotension. Hypotension is the most frequently encountered complication of hemodialysis in children. It varies in severity, ranging from transient to severe. The incidence ranges from 20% to 30%, and patients often manifest with the sudden onset of nausea, vomiting, abdominal cramping, and tachycardia.

Hypotension may result from excessive blood loss (internal or external), rapid ultrafiltration, autonomic nervous system dysfunction, or acetate use.

Hypotension can be prevented by limiting intradialytic weight loss to less than 5% of body weight and slowing the rate of fluid exchange during dialysis.

Muscle Cramps. Muscle cramps are another frequent complication of hemodialysis. Although the pathogenesis of this condition remains unclear, three predisposing factors have been implicated:

- Hemodialysis induced hypotension;
- Rapid and excessive ultrafiltration below the child's dry weight; and
- Use of sodium poor dialysate (muscle cramps appear twice as frequently in patients dialyzed against a solution with a sodium concentration of 132 vs 145 mEq/L).

Treatment consists of volume expansion with hypertonic saline (17.5% or 3 mol/liter), 50% glucose (25% glucose in children under 30 kg), or sequential ultrafiltration. Oral quinine sulfate has been shown to reduce both the frequency and severity of muscle cramps in a double-blind study, and is effective in patients who have persistent cramping post-dialysis.

Dialysis Dysequilibrium Syndrome (DDS). DDS is a spectrum of systemic and neurologic symptoms that can occur during or post-dialysis. Initial symptoms include restlessness, headache, nausea, and vomiting. Subsequently, muscle twitching, hypertension, disorientation, and myoclonic seizures may occur and lead to life-threatening complications such as seizures, coma, and cardiac arrhythmias if left untreated.[39]

EEG features include loss of alpha wave, bursts of delta waves, and slow wave activities.

Although the pathophysiology is still unclear, the most plausible explanation is that the rapid correction of uremia leads to the development of acute cerebral edema. Patients prone to develop DDS are patients receiving their first dialysis sessions with newly diagnosed CRF in whom aggressive dialysis clearances are used. Other suggested mechanisms include rapid lowering of serum sodium, acute changes in the pH of the cerebral spinal fluid, dialysis with a low glucose dialysate, and a high rate of removal of blood urea vs. brain urea.

Prevention of DDS is preferable to the actual treatment of DDS, and is easily achieved by simple technical maneuvers.

When severe symptoms develop, dialysis should be discontinued and diagnostic evaluation (i.e., EEG) should be performed to rule out another etiology. The patient should be admitted, and pediatric nephrologist consultation is advisable.

Renal Transplant Patients

Specific emergencies that may occur following renal transplant include fever and transplant rejection. The transplanted kidney may originate from a related living donor (or identical twin 18 years or older), an unrelated living donor, or a cadaveric donor. Among these options, live-related donation among immediate family members appears to achieve better long-term renal function than the others, with minimal long-term risks to the donor.

Rejection. Renal transplantation is the preferred treatment for children with chronic renal failure over dialysis for several reasons:[40]

1. It allows an unrestricted lifestyle and more normal quality of life, including growth and development, and maximum cognitive and physical development.
2. It improves patient survival. Current five-year graft survival is 70% in the first year and continues to increase with the advent of new and less toxic immune suppressive drugs.
3. It is cost effective.

There are three types of rejection: acute, sub-acute, and chronic rejection.

Pre-operative evaluation for both living or cadeveric donors include a donor-recipient cross-match of the donor's lymphocytes against the recipient to identify preformed cytotoxic antibodies.

An acute rejection occurs when preformed cytotoxic antibodies against ABO or HLA antigens, due to a typing cross-mismatching. Rejection may be immediate or late, occurring as long as 3-4 years post-transplantation. Clinical signs include fever, malaise, anorexia, hypertension, abdominal pain, or steady increase in BUN and creatinine, and renal biopsy will show evidence of rejection. In this setting, the EP should admit the patient and obtain a nephrology consult.

Immunosuppressive drugs are usually prescribed for the life of the transplanted graft to prevent rejections and include a combination of Prednisone, Azathioprine, and Cyclosporine given in doses based on the child's weight.

Acute rejection itself can often be halted with a higher dose of intravenous methylprednisone and OKT3, which can lead to preservation of renal function.

Repeated acute rejection episodes can produce sub-acute or chronic rejection, which also may be caused by a chronic humoral rejection. This later condition is usually refractory to anti-rejection therapy and eventually leads to graft failure.

Other transplant-related complications include renal arterial

and venous stenosis, thromboembolism, osteonecrosis, medication side effects (i.e., hypertension from cyclosporine), and recurrence of original disease in the grafted kidney.

In summary, prevention of long-term and late complications of renal transplant are essential and begin during the planning phase of medical management of children with CRF.

The five-year success rate of kidney transplants in children approaches that for adults, and shorter duration and less toxic drugs should improve the ability to prevent acute and chronic rejection.[41]

Fever. Fever is not an uncommon event after and during renal transplants. Known causes of febrile episodes are multiple: [42]

- Defective granulocytic function seen in CRF;
- Impaired cellular immune function;
- Drugs used before, during, or after transplant (i.e. Bleomycin);
- Serious-illness such as sepsis, rejection, pneumonia, meningitis; and
- Minor, incidental illness.

Regardless of the causes, the final pathway is the genesis of endogenous pyrogen which alter the hypothalamus heat regulatory set-point and result in heat generation and conservation. Fever is also a part of inflammatory response produced by cytokin-mediated host defense mechanisms.

True fever is accompanied by tachycardia and sweating. Ancillary tests are useful in establishing the etiology of the fever and include CBC, blood cultures, radiographs of the chest, and in appropriate situations, renal biopsy.

The EP should act promptly to hospitalize toxic-appearing children for diagnostic evaluation, antibiotic administration, and close observation. Antipyretics, such as acetaminophen, are indicated to control fever after a complete evaluation is done. Ibuprofen is to be avoided. Parenteral antibiotics should be given early to high-risk children ages 2-24 months who display signs of bacterial infection. Continuation of antibiotic therapy will be guided by results of appropriate cultures.

Conclusion

In summary, emergencies in children with CRF, although an infrequent event, represent a special entity requiring comprehensive diagnostic and decision-making skills.

The unique presentations of these true emergencies may be due to reversible causes which should be identified and treated. Life-threatening conditions should be recognized and aggressively managed. Consultation with the pediatric nephrologist is advised before final disposition of the patient in the ED.

References

1. Foreman JW, Chan JCM. Chronic renal failure in infants and children. *J Pediatr* 1988;113:793-799.
2. Kaplan BS, Kaplan P, Ruchelli E. Inherited and congenital malformations of the kidneys in neonatal period. *Clin Perinatol* 1992; 19:197-211.
3. Moxey-Mims M. Renal tubular disoders in the Neonate. *Clin Perinatol* 1992;19:159-178.
4. Kashtan CE. Alport syndrome, basement membrane and collagen. *Pediatr Nephrol* 1990;4:523-527.
5. Ritz E, Bundschu HD, Massry SG. Chronic Renal Insufficiency. In: Massry SG, Glassock RJ, eds. *Textbook of Nephrology, Volume II* (3rd Ed.). Baltimore: Williams and Wilkins; 1995:1474-1481.
6. Knight F, Gorynski L, Bentson M, et al. Hemodialysis of the infant or small child with chronic renal failure. ANNA Journal 1993; 20:315-323.
7. Leichter HE, Kher KK. Management of End-Stage Renal Failure. Dialysis Therapy. In: Kher KK and Makkur SP, eds. *Clinical Pediatric Nephrology*. New York: McGraw-Hill; 1992:559-593.
8. Evans ED, Greenbaum, LA, Ettenger, RB. Principles of renal replacement therapy in children. *Pediatr Nephrol* 1995;42: 1579-1596.
9. Maher JF. Physiology of the peritoneum: Implication for peritoneal dialysis. *Med Clin North Am* 1990;74:985-996.
10. Alexander SR. Peritoneal Dialysis. In: Holliday M, Barratt MT, Avner ED, eds. *Pediatric Nephrology, Ed. 3*, Baltimore: Williams and Wilkins; 1994:1339-1353.
11. Bishop NA, Welch TR, Strife CF. Continuous hemofiltration in children. *Pediatrics* 1990;85:819-824.
12. Bunchman TE. Continuous arterio-venous hemofiltration in infants and children. *Pediatr Nephrol* 1994;8:96-100.
13. Harmon WE, Jabs K. Hemodialysis in Children. In: Holliday M, Barratt MT, Avner ED, eds. *Pediatric Nephrology*. 1994:1354-1372.
14. Port FK. Mortality and causes of death in patients with end-state renal failure. *Am J Kidney Dis* 1990;15:215-225.
15. Brem AS. Disorders of K^+ homeostasis. *Pediatr Clin North Am* 1990;37:419-440.
16. Morris KP, Skinner JR. Cardiac abnormalities in chronic renal failure. *Arch Dis Child* 1993;68:637-643.
17. Blumberg A, Wiedmann P, Ferrari P. Effect of prolonged bicarbonate administration on plasma K in terminal renal failure. *Kidney Int* 1992;41:369-374.
18. Kemper MJ, Harps E, Muller-Wiefel DE. Hyperkalemia: Therapeutic options in acute and chronic renal failure. *Clin Nephrol* 1996; 46:67-69.
19. McClure RJ, Prasad VK, Brocklebank JT. Treatment of hyperkalemia using IV and nebulized salbutamol. *Arch Dis Child* 1994;70: 126-128.
20. Allon M, Copkney C. Albuterol and insulin for the treatment of hyperkalemia in hemodialysis patients. *Kidney Int* 1990;38:869-872.
21. Murdoch IA. Treatment of hyperkalemia with IV salbutamol. *Arch Dis Child* 1991:66:527-528.
22. Chang LH, Wu SC, Huang TP. Hypokalemic effects of IV or nebulization of salbutamol in patients with CRF. *Am J Kidney Dis* 1994;23:266-271.
23. Berry PL, Belsha CW. Hyponatremia. *Pediatr Clin North Am* 1990; 37: 351-358.
24. Conley SB. Hypernatremia. *Pediatr Clin North Am* 1990;37: 257-260.
25. Brewer ED. Disorders of Acid-Base. *Pediatr Clin North Am* 1990;37:429-440.
26. Hanna HD, Chan JCM, Gill JR Jr. Hypertension and the kidney. *J Pediatr* 1991;118; 327-341.
27. From Task Force on BP Control in Children: Report of the Second Task Force on Blood. *Pediatrics* 1987;79:1-25.
28. Calhoun DA, Oparil S. Treatment of Hypertensive Crisis. *N Engl J Med* 1990;323:1177-1183.
29. Cetta F. Malignant hypertension. *J Pediatr* 1991;118:981-986.
30. Leehey DJ. Peritonitis and Exit-Site Infection. In: Daugirdas JT, Ing TS, eds. *Handbook of Dialysis*. Boston: Little Brown; 1994:338-362.
31. Levy M, Balfe JW. Optimal approach to the prevention and treat-

ment of peritonitis in children undergoing CAPD and CCPD. *Seminars in Dialysis* 1994;7:442-449.
32. Port FK. Risk of peritonitis and technique failure by CAPD connection technique: A national study. *Kidney Int* 1992;42: 967-974.
33. Michael G. Fungal peritonitis in patients on peritoneal dialysis. *Am J Nephrol* 1994;14:113-20.
34. Hakim RM, Depner TA. Adequacy of dialysis. *Am J Kidney Dis* 1992;20:107-123.
35. Fine RN. Dialysis in Infants and Children. In: Daugirdas JT, Ing TS, eds. *Handbook of Dialysis*. Boston: Little Brown; 1994:553-568.
36. Fan PY, Schuab SJ. Vascular access: Concepts for the 1990s. *J Am Soc Nephrol* 1992; 3:1-10.
37. Bregman H, Daugirdas JT, Ing TS. Complications During Hemodialysis. In: Daugirdas JT, Ing TS, eds. *Handbook of Dialysis*, 2nd ed. Boston: Little Brown; 1994:149-168.
38. Jameson MD, Wiegmann TB. Principles, uses and complications of hemodialysis. *Med Clin North Am* 1990;74:945-959.
39. Warady BA. The use of the peritoneal equilibration test to modify peritoneal dialysis modality in children. *Seminars in Dialysis* 1994;7: 403-408.
40. Meyer KE, Weiland H, Thomson PD. Pediatric renal transplant noncompliance. *Pediatr Nephrol* 1995;9:189-192.
41. Welch TR. Treatment options for end-stage renal disease in children. *Clin Trans* 1990;5:146-149.
42. McKay DB, Milford EL, Seyegh MH. Clinical Aspects of Renal Transplant. In: Brenner BM, ed. *The Kidney*, 5th Ed., Vol. 2. Philadelphia: W.B. Saunders; 1996:553-568.

Physician CME Questions

1. The decline in the kidney's filtering capacity in children with CRF does *not* result in volume overload unless GFR is:
   A. less than 5% of normal for age.
   B. less than 10% of normal for age.
   C. less than 15% of normal for age.
   D. less than 20% of normal for age.
   E. None of the above

2. Pediatric patients with CRF usually display a decreased capacity for excreting potassium when the GFR is:
   A. less than 5% of normal for age.
   B. less than 10% of normal for age.
   C. less than 15% of normal for age.
   D. less than 20% of normal for age.
   E. None of the above

3. All of the following ECG changes are usually due to hyperkalemia *except:*
   A. peaked T waves.
   B. widening of QRS.
   C. flattened P waves.
   D. supraventricular tachycardia.
   E. ventricular tachycardia.

4. The drug(s) of choice for treating cardiac toxicity due to hyperkalemia in children with CRF is (are):
   A. polysterene sulfonate (Kayexalate).
   B. intravenous sodium bicarbonate.
   C. intravenous glucose and insulin.
   D. intravenous calcium.
   E. All of the above

5. According to the 1987 report from the National Heart, Lung, and Blood Institute, significant hypertension as systolic or diastolic in children is defined as:
   A. less than 90th percentile of the standard norm for age and sex.
   B. between 90th percentile and 95th percentile of the standard norm for age and sex.
   C. between 95th percentile and 99th percentile of the standard norm for age and sex.
   D. above 99th percentile of the standard norm for age and sex.

6. The most effective means of reducing intra-vascular volume in children with CRF is:
   A. high flow oxygen.
   B. intravenous nitroglycerin.
   C. emergent dialysis.
   D. intravenous diuretics.
   E. intravenous nitroprusside.

7. In children younger than 5 years, the most common cause of CRF is:
   A. acquired diseases.
   B. hereditary diseases.
   C. congenital diseases.
   D. metabolic diseases.
   E. endocrine etiologies.

8. Chronic renal failure in children is defined as progressive reduction in GFR to:
   A. below 25% of normal value for at least 12 months.
   B. below 25% of normal value for at least 6 months.
   C. below 25% of normal value for at least 3 months.
   D. below 10% of normal value for at least 6 months.
   E. below 10% of normal value for at least 3 months.

9. Which of the following is a sign of peritonitis in a child who has CHF and is on dialysis?
   A. Fever
   B. Vomiting
   C. Abdominal pain
   D. Diarrhea
   E. All of the above

10. What is the most common etiology of peritonitis in a child on dialysis?
    A. Proteus
    B. *E. coli*
    C. Staphylococcus
    D. Group A strep
    E. Fungus

In Future Issues: Submersion Injuries