Chronic kidney disease (CKD) is a complex medical condition that can be difficult to understand. The ultimate end of CKD is end-stage renal disease, which typically results in patients requiring the life-sustaining treatment of dialysis. Dialysis is a complicated, life-altering and expensive process. This article explores the pathophysiology of CKD, the process of dialysis and the complications commonly encountered by EMS providers treating and transporting these patients.

Incidence

Kidney disease is a serious and expanding problem: end-stage renal disease has a three-year mortality of greater than 50%. More than 10% of the U.S. population has some sort of chronic kidney disease. Chronic kidney disease is the progressive and irreversible loss of the kidneys’ ability to filter wastes and toxins from the blood. Kidney function is measured by the glomerular filtration rate (GFR): the amount of blood filtered by the nephron’s glomeruli in a minute. Normal GFR is greater than 90 mL/min/1.73m². In the U.S., CKD is classified into five stages, beginning with mild damage for which the body can compensate and ending with end-stage renal disease (ESRD), defined by a GFR of less than 15 mL/min and the need for chronic renal replacement therapy (RRT). In 2010, 114,000 people began dialysis, and nearly 3,000 more received kidney transplants as their initial ESRD intervention.1

The only definitive treatment for ESRD, a kidney transplant, is not available for many patients. More than 95,000 individuals currently await kidney transplants. The increasing number of patients receiving hemodialysis helps demonstrate that the overall
number of kidney transplants is quite low compared to the actual need. At the end of 2010, there were more than 400,000 patients on dialysis and only 180,000 living with kidney transplants.

While the many causes for renal failure will be discussed later in this article, diabetes is by far the leading cause of ESRD, and its incidence has been increasing since 1996. Together, diabetes and hypertension are the primary causes of kidney disease in more than 70% of cases. \(^1\)

Normal Renal Physiology

The kidney is a bean-shaped organ found in the retroperitoneal region. It is located just lateral to the vertebral column between the 12th thoracic and third lumbar vertebra. Typically the right kidney is situated slightly inferior compared to the left because of the liver (Figure 1). Like all solid organs, the kidney is enclosed in a fibrous capsule that provides protection.

Blood enters the kidney through the renal artery at the hilum, which is the deep slit in the kidney’s medial aspect. The continuous pale outer tissues of the kidney are known as the renal cortex, which surrounds the inner renal medulla. The shape and interaction of the renal medulla and cortex cause the medulla to take on triangular shapes known as renal pyramids.

Found within the renal cortex and medulla is the nephron, the kidney’s microscopic functioning unit. The nephron serves as the body’s filter and removes toxins and wastes from the blood. Each kidney has roughly 1.25 million nephrons that total a length of more than 145 km. The nephron (Figure 1A) has two primary structures: the renal corpuscle and the renal tubule. The renal corpuscle (in the cortex) filters blood at the glomerulus into Bowman’s capsule. The glomerulus is a capillary network and is enveloped by Bowman’s capsule, which is the start of the nephron’s waste collection system. The two are separated by a thin epithelial wall. It is within the glomerulus where the blood pressure forces fluid and dissolved solutes out of an arteriole and into Bowman’s capsule. The fluid and solutes forced from the blood are called filtrate and drain into the proximal convoluted tubule. Damage to the barrier between the glomerulus and Bowman’s capsule will lead to CKD.

Once in the proximal convoluted tubule, filtrate drops down into the loop of Henle, where the body reabsorbs sodium, potassium and roughly 90% of the fluid within the filtrate. This concentrates the filtrate; at the same time, additional wastes are added. Filtrate then leaves the loop of Henle and enters the distal renal tubule, where the body excretes toxins, acids, drugs and free ions. Additional sodium is also reabsorbed. Several renal tubules combine into a collection duct, at which point the filtrate is considered urine. The collection ducts continue to grow in size as more tubules combine, and all of the ducts drain into what eventually becomes the ureter and then the bladder. A healthy individual produces 0.5–1 mL/kg/hr of urine in a day. When patients progress toward renal failure, there are different terms for the amount of urine they produce (Table 1).

### Table 1: Urine output In Renal Failure

<table>
<thead>
<tr>
<th>Type</th>
<th>Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0.5-1mL/kg/hr</td>
</tr>
<tr>
<td>Non-oliguria</td>
<td>&gt;400mL/day</td>
</tr>
<tr>
<td>Oliguria</td>
<td>50-400mL/day</td>
</tr>
<tr>
<td>Anuria</td>
<td>&lt;50mL/day</td>
</tr>
</tbody>
</table>

### Acute Renal Failure

It’s important to understand the difference between acute renal failure (ARF) and chronic renal failure (CRF). More than 50 pathologies can lead to ARF; some of the most common are identified in Table 2. ARF is recognized through a rapid decline in renal function and urine output at a rate measured in hours or days. During this decline the patient’s body quickly loses homeostasis as toxins accumulate and electrolyte balances are lost. ARF is
a serious emergency with a high mortality.

There are three types of ARF, which are named to describe the location of the pathology. **Prerenal** failure results from renal hypoperfusion, which can occur for a variety of reasons such as hypotension or sepsis. **Intrarenal** failure indicates injury or pathology affecting the nephron or renal parenchyma, and **post renal** failure generally suggests an obstructive pathology in the drainage system. Patients with severe acute renal failure may briefly require dialysis, but otherwise the treatment of ARF focuses on correcting the underlying cause. ARF cannot be corrected until the actual cause is identified. Without intervention ARF can cause chronic renal failure. It is not uncommon for patients with CRF to have acute-on-chronic renal failure from isolated events such as an obstructive renal stone or infection. It is beyond the scope of this article to identify all of the causes of ARF, as most cannot be managed in the prehospital environment.

### Chronic Renal Failure

While ARF is a true medical emergency with a rapid onset, its causes are reversible and often transient in nature. CRF is life-altering and permanent once it develops. There are many causes of chronic renal failure, which are outlined in Table 3. Of note, children can develop CRF as a result of congenital abnormalities or diseases such as sickle cell anemia or following traumatic injury.

The diagnosis of CKD is determined based on a patient’s estimated glomerular filtration rate (eGFR). A normal GFR, or the amount of blood passing through glomeruli in a minute, is greater than 90 mL/min/1.73m². Diagnosis of CKD is defined as: kidney damage for three months or more as indicated by structural or functional abnormalities of the kidney, with or without decreased GFR, or a GFR less than 60 mL/min for three months or more, with or without kidney damage. CKD progresses through stages 1–5 based on the eGFR. Stages 1–3 have a GFR greater than 30, and patients are often asymptomatic. Typically patients become symptomatic with a GFR less than 30 mL/min (stage 4). CKD has an impact on nearly every system in the body; many laboratory values become abnormal as kidney function worsens. Table 4 lists the normal ranges for some of the lab values often altered in patients with CKD. When patients reach stage 5, their GFR is less than 15 mL/min/1.73m², and dialysis is indicated.

Patients do not commonly go directly from healthy to a diagnosis of CKD. With the exception of situations such as major trauma, a patient will typically experience a progression of renal dysfunction as they move toward renal failure. As a patient’s GFR decreases, azotemia, the accumulation of nitrogen and other wastes usually removed through urine within the blood, begins to occur. As the patient’s failure to excrete wastes via kidney progresses, the concentration of urea (byproduct of protein breakdown) rises, and a more severe condition develops: uremia. Simultaneously, the hormones released by the kidneys decrease in blood concentration as kidney function declines. These hormones include erythropoietin, which controls red blood cell production, a lack of which causes anemia. Decreased production of another hormone, renin, disrupts the renin-angiotensin-aldosterone system (RAAS), contributing to the often-severe hypertension observed in these patients.

### Life With CRF and Dialysis

The definitive treatment for end-stage renal disease is a kidney transplant. Unfortunately, transplant is only available for a small percentage of the population; annually fewer than 20,000 transplants are performed in the United States. Without intervention ARF can cause chronic renal failure. It is not uncommon for patients with CRF to have acute-on-chronic renal failure from isolated events such as an obstructive renal stone or infection. It is beyond the scope of this article to identify all of the causes of ARF, as most cannot be managed in the prehospital environment.

### Table 2: Causes of Acute Renal Failure to Ask

<table>
<thead>
<tr>
<th>Type</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal</td>
<td>renal hypoperfusion (hypovolemia, hypotension)</td>
</tr>
<tr>
<td>Intrarenal</td>
<td>Rhabdomyolysis, infection</td>
</tr>
<tr>
<td>Post renal</td>
<td>kidney stones, tumors</td>
</tr>
</tbody>
</table>

### Table 3: Causes of Chronic Renal Failure

<table>
<thead>
<tr>
<th>Pathologic</th>
<th>Physiologic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glomerulonephritis</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Interstitial nephritis</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Tumors</td>
<td>Chronic urinary tract infections</td>
</tr>
<tr>
<td>Transplant rejection</td>
<td>Congenital abnormalities</td>
</tr>
<tr>
<td>Congenital disease</td>
<td>Vascular disease</td>
</tr>
<tr>
<td></td>
<td>HIV</td>
</tr>
</tbody>
</table>

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All other patients with ESRD will receive dialysis treatment several (typically three) times a week. Dialysis is an intervention that does nothing to treat the cause of ESRD; rather, it temporarily corrects the effects of poorly functioning kidneys. Dialysis removes excess fluids and electrolytes, toxins (urea) and other wastes no longer eliminated effectively. There are two forms of dialysis: peritoneal dialysis and hemodialysis. The vast majority of patients receive hemodialysis either in a hospital or at one of the thousands of outpatient dialysis centers across the country.

Table 4: Normal Ranges for Lab Values Commonly Effected by CRF

<table>
<thead>
<tr>
<th>Lab</th>
<th>Normal Range</th>
<th>Effect in CRF</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN</td>
<td>8–25mg/dl</td>
<td>elevated</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.6–1.2 mg/dl</td>
<td>elevated</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5–5 mEq/L</td>
<td>elevated</td>
</tr>
<tr>
<td>Sodium</td>
<td>135–145</td>
<td>decreased</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>22–26</td>
<td>decreased</td>
</tr>
<tr>
<td>pH</td>
<td>7.35–7.45</td>
<td>increased</td>
</tr>
</tbody>
</table>

Placing a patient on dialysis fundamentally changes their life and marks a transition in their health from a regular and constant body cleansing of toxins to a lifestyle where there is recurrent buildup followed by rapid removal. The result is significant effects on nearly every organ system of the body.

The cardiovascular system is affected in several ways. Chronic anemia develops and results in chronic fatigue, reduced physical capacity to perform daily functions, impaired immune function and cognition, reduced quality of life, increased cardiovascular disease and increased mortality. A normal hemoglobin is roughly 15 g/dL; in 2010, 10% of ESRD patients experienced a hemoglobin of less than 10 g/dL, and only 21.7% had hemoglobin greater than 12 g/dL. Platelets are also stressed and prone to damage during hemodialysis.

Overall heart disease is the leading cause of death for ESRD patients; heart failure, left ventricular hypertrophy, arteriosclerosis and stroke are all common.

Hypertension, if not already a contributing cause of the renal failure, becomes a significant problem for patients as their ability to regulate the RAAS progresses. This is further exacerbated by the body’s inability to excrete sodium and fluid effectively. In addition to sodium balance concerns, patients with ESRD also need careful potassium balance; hyperkalemia is a common problem. The risk for experiencing hyperkalemia is exacerbated by the body’s increased catabolism (cell destruction), chronic acidosis from waste accumulation, and potassium-based medicines.

The normal potassium range is 3.5–5 mEq/L, and levels become dangerous above 6 mEq/L. Advanced providers may see the earliest signs of hyperkalemia on an EKG by the presence of peaked
T-waves. A T-wave at least two-thirds the height of the QRS complex is considered peaked and is typically present in all leads. If potassium levels exceed 8 mEq/L, the P-wave is lost, and the QRS begins to widen. Eventually the S and T waves begin to slope together into what is considered a sine wave. Management of hyperkalemia is discussed later.

The last major cardiovascular problem patients with ESRD risk experiencing is pericarditis. Pericarditis may develop as a result of high uremic acid levels but may also develop if dialysis is inadequate and does not remove enough fluid or acids. When these conditions occur, high uremic levels (usually BUN is greater than 60 mg/dL) cause inflammation of the pericardial sac. Pericarditis causes chest pain (often relieved by the tripod position) and ST segment changes in all areas of a 12-lead EKG, and may cause a fever. Listening to heart tones may reveal a friction rub. Maintain a high index of suspicion for pericarditis whenever patients with ESRD complain of chest pain, as up to 20% experience it at some point.

The nervous system is also affected by ESRD as neurons are impaired and damaged, though the etiology of why is unclear. In the central nervous system, uremia-related dementia can occur. Peripherally, neuropathy and restless leg syndrome are common.7

Pulmonary edema is also common, especially when patients miss dialysis or consume too much water or sodium. Additionally, patients are at risk for experiencing pulmonary effusions. Tachypnea is common and should almost be anticipated in patients with ESRD as the body tries to compensate for a stressed cardiovascular system, and fast breathing also blows off extra carbon dioxide as a buffer mechanism for the presence of increased acids.

In the gastrointestinal system, the constant fluctuation of fluid accumulation and then dumping by the body leads to regular bloating and cramping as built-up fluids third-space into the interstitial spaces of the GI tract. Patients often experience nausea, vomiting and chronic diarrhea, making malnutrition common.

Inspect the skin for uremic frost as a sign of renal failure. It looks like a sandy or salty frost and is commonly seen in the eyelashes, lips, armpits and groin. Additionally, the skin is affected by chronic dryness, cracking and loss of elasticity.7

Dialysis
Dialysis works by diffusion by placing a semipermeable membrane between the blood and dialysate (dialysis solution). Keep in mind that the patient’s blood contains no sodium or potassium; it is concentrated with other metabolites too large to diffuse into the body. The absence of sodium and potassium allows both electrolytes to diffuse into dialysate.

Peritoneal dialysis introduces dialysate through an implanted catheter and into the patient’s peritoneal cavity, where it is left for several hours. In this case the body acts as the semipermeable membrane. After several hours of the

“When hyperkalemia is suspected, look for profound EKG changes, including peaked T waves, loss of a P wave and a widened QRS complex.”
patient carrying dialysate in the abdomen, it is removed.

Hemodialysis removes the blood from the body via an access site and pulls it into the dialysis machine, which contains a semipermeable membrane. The dialysis machine circulates dialysate past the blood at a rate of 500 mL/min, which very rapidly pulls excess wastes, fluid and electrolytes from the bloodstream. Hemodialysis is much faster than peritoneal dialysis, but it is less comfortable for patients, as there are relatively fast fluid and electrolyte shifts to which the body must react. Transient hypotension frequently occurs and can cause cramping, nausea, headache, syncope, chest pain and fatigue. Generally a small fluid bolus is safe and can help the patient feel better.

It is not feasible to perform dialysis through traditional IV catheters, and if it were, the repetitive access would quickly cause a patient’s veins to become fibrous and difficult to cannulate. For patients who require long-term dialysis, vascular surgeons typically insert one of three types of access ports. The fastest access port, often used when patients first or emergently need dialysis, is a centrally placed venous line with multiple lumens. Prehospital providers may observe patients with central access ports when patients are waiting for grafts or fistulas to be implanted or ready for use. When a patient leaves a hospital with a central access port, it is most commonly in the chest, in the right or left subclavian vein. These ports have two catheters sticking out of the chest that may be covered with gauze. If you see these ports, do not access them, as it requires strict sterile technique and there is a high risk for infection. One exception to this rule is during cardiac arrest, when it is reasonable to access these sites for rapid access to the circulatory system.

Arteriovenous grafts are artificial lumens inserted in the patient’s arm that connect a vein and an artery. While grafts are thought of as temporary devices, they may be used for several years. Like any foreign item in the body, a graft is prone to infection; their risk is particularly high, as they are regularly punctured and accessed. Additionally, grafts are at risk for clotting.

The most permanent dialysis site is a fistula. A fistula connects an artery and a vein together and is surgically created in the upper or lower arm. Connecting the two increases the diameter of the new “vessel,” making access easier. This increases the strength of the vessel’s wall and allows an increased flow to support blood removal without inhibiting limb perfusion during dialysis. Traditionally, EMS providers are advised to not access fistulas, as they are not true venous access sites and improper access may damage them.

Management Considerations

Dialysis patients experience several common complications prehospital
providers should be prepared to manage. They also require special considerations for management in general.

Any time you manage a patient with ESRD who complains of any illness, fatigue or fever, ask what site is used for their dialysis (if they receive it) and inspect it for signs of infection (redness, localized pain, warmth and/or pus around any wound or puncture site). Dialysis access sites are common infection sources, and SIRS and sepsis can rapidly develop. Special consideration is needed for patients being treated with peritoneal dialysis, as they are at an increased risk for peritonitis.

During dialysis patients receive heparin to prevent clots from developing at the access site. Further, they are chronically coagulopathic and as a result may experience bleeding from their graft or fistula when the access catheters are removed. Bleeding from these sites may appear profuse but is easily controlled with well-aimed direct pressure on the small hole at the puncture site. Consider placing a rolled piece of gauze over the puncture and applying moderate pressure with your hand or a compressive dressing. If bleeding continues, the pressure is not adequate, and the bondage needs to be more directly applied. Do not simply add more bandages over the top, as these only serve to absorb more blood. Once bleeding is controlled, pressure may be required for 30–60 minutes for a clot to completely form. When requested to treat a patient with postdialysis bleeding, anticipate needing to maintain pressure on the bleeding site for the duration of the transport to the hospital.

Following dialysis patients may experience dizziness and hypotension. In some cases more than six liters of fluid are removed from these patients’ bodies. Following dialysis, judicious fluid boluses with normal saline are reasonable, but proceed cautiously, as the hypotension is most likely transient. Administer fluid to ESRD patients between dialysis appointments with caution and only after assessing for symptoms of dehydration or fluid overload. Dry skin, flat neck veins and dry mucous membranes suggest dehydration, while bulging neck veins, crackles on auscultation, peripheral edema and complaints of bloating indicate fluid overload. A fluid bolus is appropriate in the former but should be avoided in the latter.

Patients with histories of missed dialysis or medication noncompliance are at risk for experiencing metabolic acidosis and electrolyte imbalances such as hyperkalemia. Consider presumptive treatment of these two conditions with any dialysis patient in cardiac arrest. This would include sodium bicarbonate (1 mEq/kg) for acidosis and calcium chloride (500 mg–1 g) for hyperkalemia. Proceed in accordance with local protocols; if this is not in yours, ask your medical director about it.

IV access in the dialysis patient can be a challenge. Always attempt to establish peripheral access in a limb without a fistula or graft. If necessary, use the limb with an access site. There is debate over whether to access a graft or fistula during medical emergencies. The overall high infection and complication rate makes it reasonable to avoid accessing fistulas and grafts, and it should not be done without online medical direction. During cases such as cardiac or respiratory arrest or when a patient is in a critical condition, a fistula can be easily cannulated with a traditional IV needle; guidelines for this should be provided in local protocols.

Impaired renal function affects medication metabolism and clearance time, so caution is advised with any medication administration. There are frequently questions regarding the effectiveness of furosemide in ESRD patients. Furosemide has been shown to be beneficial in reducing left ventricular preload through venous dilation within 15 minutes of administration in CKD patients. A 1992 study found that small doses of oral furosemide temporarily increased urine output over a 24-hour period without serious complications. But in a more recent study on ESRD patients undergoing peritoneal dialysis, furosemide had no effect on increasing urine output.

What does this conflicting research suggest? There is a need for more data, and the decision to administer will be case-based and made in consultation with medical control. If there is pulmonary edema, consider vasodilating drugs as well as noninvasive positive-pressure ventilation.

Conclusion

Chronic kidney disease is a worsening epidemic in the United States and is complicated by many associated problems. When a patient develops ESRD, the only options for continued life are a kidney transplant or dialysis. Hemodialysis must be performed three times a week to maintain homeostasis within the body and eliminate toxins and metabolic wastes.

Approach ESRD patient management carefully. These are complicated patients at high risk for cardiovascular and pulmonary emergencies. Prehospital interventions can manage symptoms, but transport to dialysis or an emergency department is generally necessary.

REFERENCES


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