Acute Renal Failure

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Objectives

By the end of this learning experience, the clinician will be able to define Acute Renal Failure, discuss the three classifications of Acute Renal Failure and describe the four phases of Acute Renal Failure.

Introduction

Acute renal failure (ARF) or Acute Kidney Injury (AKI as it is now sometimes called in medical literature) has in the past had a very generic definition that generally consisted of: the abrupt decrease in renal function that occurs over hours to days. However, this ambiguity has caused much debate and confusion. Recently the Acute Dialysis Quality Initiative (ADQI) adopted some guidelines to the treatment of ARF similar to the gold standard in chronic renal failure of the National Kidney Foundation DOQI guidelines. The official definition called RIFLE adopted by ADQI is multifactoral and consists of the following components:

- "Risk (R) - Increase in serum creatinine level X 1.5 or decrease in GFR by 25%, or UO <0.5 mL/kg/h for 6 hours
- Injury (I) - Increase in serum creatinine level X 2.0 or decrease in GFR by 50%, or UO <0.5 mL/kg/h for 12 hours
- Failure (F) - Increase in serum creatinine level X 3.0, decrease in GFR by 75%, or serum creatinine level $\geq$ 4 mg/dL; UO <0.3 mL/kg/h for 24 hours, or anuria for 12 hours
- Loss (L) - Persistent ARF, complete loss of kidney function > 4 wk
- End-stage kidney disease (E) - Loss of kidney function > 3 months" (Sinert).

Acute Renal Failure is a common entity in the emergency room; and emergency room clinicians can play a pivotal role in the early recognition of this life-threatening event, reversing the cause of the destruction, and placing the patient on the path to return of renal function. It is important to recognize that there is a distinction between community acquired ARF and hospital acquired ARF. Hospital acquired ARF is most commonly seen in the ICU setting and is frequently a consequence of medical procedures or multi-system organ failure. This type of ARF has the highest mortality rate and most often is the type that progresses to End Stage Renal Disease. Community acquired ARF is most often the result of volume depletion, which is usually and easily correctible situation and viable renal function is restored in up to 90% of patients. Mortality rates are lower in patients not requiring dialysis and patients that do not become oliguric during their course of ARF.

"The clinical condition of acute renal failure (ARF) is said to occur in anywhere from 1 to 25% of critically ill patients depending on the population being studied and the criteria used to define its presence. Furthermore, mortality in these populations ranges from 28 to 90%" (ADQI 2,1., 2003).

The disease is usually manifested by elevated levels of blood urea nitrogen (BUN) and creatinine. Creatinine levels that are elevated to above 50% of baseline or increased 0.5mg/dL indicate acute renal failure. However, immediately after an insult to the kidney, BUN and creatinine levels may remain normal and the only symptom present may be diminished urine output. In addition, alterations in electrolyte balance, such as sodium and potassium and alterations in acid-base balance may occur. The most common causes are any condition that may interrupt the blood flow to the kidney such as acute hypotension, dehydration, or cardiac arrest, and direct toxic effects to the kidneys as from medications or chemicals.

AKF may occur in 3 clinical classifications, including the following: as an adaptive response to severe volume depletion and hypotension, with structurally intact nephrons (pre-renal), in response to toxic, ischemic, or inflammatory insults to the kidney, with structural and functional damage (intrinsic) and with obstruction to the passage of urine (post renal). Pre-renal conditions account for 50-80% of all the cases of acute renal failure.

Patients commonly present with symptoms related to hypovolemia, including thirst, decreased urine output, dizziness, and orthostatic hypotension. Hypotension and tachycardia are obvious clues to decreased renal perfusion. Look for a history of excessive fluid loss via hemorrhage, GI losses, sweating, or renal sources. Insensible fluid losses can result in severe hypovolemia in patients with restricted fluid access and should be suspected in the elderly and in comatose or sedated patients. Evaluation for hypovolemia should include evaluations for orthostatic hypotension, mucosal membrane moisture, and tissue turgor.

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Prerenal AKI can also result from decreased renal perfusion in patients with heart failure or shock (eg, sepsis, anaphylaxis). Patients with advanced cardiac failure leading to depressed renal perfusion may present with orthopnea and paroxysmal nocturnal dyspnea. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are medications that are frequently used in the treatment or chronic renal failure; however, these medications can cause ARF in volume-depleted states. Radiocontrast agents, nonsteroidal anti-inflammatory drugs (NSAIDs), amphotericin, calcineurin inhibitors, norepinephrine, vasopressor medications and hypercalcemia can all cause arteriolar vasoconstriction and ARF. With intrinsic renal failure, removal of tubular toxins and initiation of therapy for glomerular diseases decreases renal afferent vasoconstriction (Sinert, 2006). Hepatorenal syndrome is considered a form of ARF due to the vasoconstriction of the blood vessels supplying the kidney.

Pre-renal disruptions in blood flow can lead to intrinsic damage if not corrected and treated early enough. The process of filtration within the kidney is dependent upon the pressure gradient from the glomerulus to the Bowman's space. This pressure gradient is supplied by the degree of perfusion within the renal vasculature. The insult begins with a period of decreased perfusion to the kidney usually with a mean arterial pressure (MAP) of less than 75mm Hg. This initiates several autoregulatory processes within the kidney to cause severe constriction of the arterioles supplying blood to the glomerulus. The adaptive mechanisms to hypoperfusion are autoregulation and release of rennin. In auto regulation, the blood flow to the glomerulus is regulated by constriction of the efferent arteriole and dilation of the afferent arteriole. This means that more blood is coming into the glomerulus than is leaving, and thus perfusion is increased. In addition, the kidneys release rennin, which activates the angiotensin-aldosterone system, which causes peripheral vasoconstriction and retention of sodium, thereby increasing blood pressure and renal perfusion. The increase in sodium also leads to the retention of water by releasing antidiuretic hormone. When these adaptive mechanisms fail to keep up with the degree of injury, ischemia results.

Structural injury in the kidney is the hallmark of intrinsic AKI, and the most common form is acute tubular necrosis (ATN), either ischemic or cytotoxic. Ischemia that lasts between 60-90 minutes causes irreversible damage within the kidney. This initial ischemic insult triggers production of oxygen free radicals and enzymes that continue to cause damage to the cells that often continues after the insult is removed. Blood flow can be reduced as much as 50%. The small blood vessels of the kidney are now more sensitive and less tolerant to changes in blood pressure and the effects of vasoconstrictive medications. These blood vessels loose the autoregulatory mechanism and the ability to vasodialate in response to decreased perfusion. Any event which causes hypotension, including hemodialysis, can cause further damage to the kidneys. Careful monitoring and assessment of blood pressure is one of the primary preventions to avoid further renal compromise. Tubular cellular damage results in disruption of tight junctions between cells, allowing back leak of glomerular filtrate and

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further depressing effective GFR. In addition, dying cells slough off into the tubules, forming obstructing casts, which further decrease GFR and lead to oliguria.

Post renal failure occurs because of some obstruction in the urinary system below the level of the kidneys. Common causes of post renal failure include kidney stones, strictures, tumors, and enlarged prostate. If the obstruction only involves one side of the renal collecting system between the renal pelvis and the bladder, serum creatinine may remain normal as the function of the opposite kidney increases to meet the body's demands. Patients who develop anuria typically have obstruction at the level of the bladder or downstream to it. Relief of urinary obstruction, results in decompression within the kidney and decreased vascular constriction restoring renal blood flow and filtration.

While these classifications are useful in differentiating the diagnosis, there is a lack of uniformity in the features of the different categories. This lack of consistency is a reflection of the diverse nature of the etiologies leading up to the injury. Rises in creatinine may be rapid or slow, but are always exponential, not linear. Therefore, each increment of elevation in creatinine is reflective of a multi-fold loss of nephron function.

Creatinine 1.0 mg/dL - Normal GFR

Creatinine 2.0 mg/dL - 50% reduction in GFR

Creatinine 4.0 mg/dL - 70–85% reduction in GFR

Creatinine 8.0 mg/dL - 90–95% reduction in GFR

Differentiating between pre-renal azotemia and ATN can be difficult. In pre-renal azotemia, urine output is diminished. In ATN, urine output may or may not be diminished. In pre-renal assaults, the urinalysis will show normal urinary sediment with hyaline or granular casts, high specific gravity, high osmolality, low urinary sodium and urea, and normal urine creatinine. The urinalysis of the ATN patient will exhibit low specific gravity, low osmolality, increased urinary sodium, low urine urea, and creatinine. Another distinguishing element is the response to therapy. In pre-renal conditions, there is no actual damage to the kidney itself, while with ATN damage to the nephrons is present. In pre-renal conditions, the kidneys will respond to therapy aimed at correcting the cause with rapid improvement in renal function while in ATN these improvements are not seen from measures such as correction of shock, dehydration, or hypotension.

Oliguria by definition is a 24-hour urine output of less than 400 ml. Oliguria in the presence of intrinsic renal damage has a poorer prognosis than if pre-renal failure is the cause. Urine output of less than 100ml/24 hr is termed anuria, and

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if abrupt in onset, signifies bi-lateral obstruction, obstruction of the bladder outlet, bladder trauma with abdominal leakage, or severe bilateral renal injury depending upon the clinical picture and presentation.

Differences in urinary composition

Pre-renal Failure	ATN
Urine specific gravity >1.018	Urine specific gravity <1.012
Urine osmolality (mOsm/kg H_2O) >500	Urine osmolality (mOsm/kg H_2O) <500
Urine sodium (mEq/L) <15-20	Urine sodium (mEq/L) >40
Plasma BUN/creatinine ratio >20	Plasma BUN/creatinine ratio <10-15
Urine/plasma creatinine ratio >40	Urine/plasma creatinine ratio <20

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Age

The patient's age has significant implications for the differential diagnosis of ARF.

Newborns and Infants

The most common cause of ARF is prerenal etiologies.

Prerenal ARF

Perinatal hemorrhage - Twin-twin transfusion, complications of amniocentesis, abruptio placenta, birth trauma

Neonatal hemorrhage - Severe intraventricular hemorrhage, adrenal hemorrhage

Perinatal asphyxia and hyaline membrane disease (newborn respiratory distress syndrome) both may result in preferential blood shunting away from kidneys (i.e., prerenal) to central circulation.

Intrinsic ARF

Acute tubular necrosis (ATN) can occur in the setting of perinatal asphyxia. ATN also has been observed secondary to medications (aminoglycosides, NSAIDs) given to the mother perinatally.

ACE inhibitors can traverse placenta, resulting in a hemodynamically mediated form of ARF.

Acute glomerulonephritis is rare and most commonly the result of maternal-fetal transfer of antibodies against the neonate's glomeruli or transfer of chronic infections (syphilis, cytomegalovirus) associated with acute glomerulonephritis.

Postrenal ARF: Congenital malformations of urinary collecting systems should be suspected.

Children

The most common cause of ARF is prerenal etiologies.

Prerenal ARF

The most common cause of hypovolemia in children is gastroenteritis.

Congenital and acquired heart diseases are also important causes of decreased renal perfusion in this age group.

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Intrinsic ARF

Hemolytic uremic syndrome (HUS) often is cited as the most common cause of ARF in children. The most common form of the disease is associated with a diarrheal prodrome caused by *Escherichia coli* 0157:H7.

These children usually present with microangiopathic anemia, thrombocytopenia, colitis, mental status changes, and renal failure.

Acute post streptococcal glomerulonephritis should be considered in any child who presents with HTN, edema, hematuria, and renal failure.

Adults

Please remember that post obstructive ARF in the elderly should never be overlooked (Sinert, 2006).

There are four phases of acute renal failure called the initiating stage, oliguric stage, diuretic stage, and recovery stage. Each phase has a different set of management principles.

In the initiating stage, which begins when the kidney is injured and lasts from hours to days, signs of renal impairment are present such as altered BUN and creatinine levels and decreased urine output. During this phase, the cause of acute renal failure is sought and treatment is initiated. The sooner that renal blood flow is reestablished the better the chance of recovering renal function with minimal damage to the nephrons. Usually this is best accomplished by restoring fluids lost through volume depletion, raising blood pressure. Complications of acute renal failure are many and may be severe or life threatening. Mortality from acute renal failure is 50% and the number one cause of death is from infections. Once the etiology of renal failure is determined, the patient needs to be informed as to the cause of the renal injury. As the disease progresses, the patient needs to be instructed about the various phases of acute renal failure and self care measures such as diet and fluid intake. The patient also needs to be informed about signs and symptoms to alert the health care provider to. Finally, during the recovery phase, the patient needs to be educated about measures to prevent further episodes of acute renal failure.

The oliguric stage is hallmarked by decrease in urine output, however, up to 50% of patients may not exhibit decreased urine output and their symptoms are less severe. Acute fluid overload may lead to compromise of a patient's ability to oxygenate and ventilate. Edema may begin to appear in dependent areas or around the face and the eye orbits. Other complications can include cardiac

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arrest from hyperkalemia due to the decrease in urine output, elevated phosphorus levels due to impaired renal regulation of calcium and phosphates, metabolic acidosis due to decreases in excretion hydrogen ions, GI bleeding, and decreased nutritional status. In treating hyperkalemia, all sources of dietary potassium should be stopped and a low potassium diet prescribed. It is important to question the patient or family about use of salt substitutes, as these may be common sources of potassium. Exchanging potassium ions across the intestinal tract using potassium-binding resins is a consideration. This may be given orally or by rectal enema. These medications usually cause diarrhea, which leads to increased potassium excretion. In addition, infusions of insulin and glucose may be used to enhance the shift of potassium into the cell. Lasix, a diuretic, can be used in an attempt to increase urine production and directly encourage to loss of potassium from the body. Double check to make sure the patient is not on a potassium sparing diuretic. Finally, dialysis may be used in the treatment of hyperkalemia. Other less serious complications can include disruptions in self-concept and sleep pattern. This stage commonly lasts from 5 to 15 days, but can last for weeks. Renal healing begins during this stage. Once blood flow is reestablished, the remaining nephrons in the kidney enlarge in an attempt to handle to increased workload caused by nephron damage.

The diuretic stage usually lasts for 1-2 weeks but can persist longer. In this stage, an increase in urine output is noted and uremia begins to resolve as the kidney continues to heal. The recovery phase is marked by completion of the healing process and the development of scar tissue in damaged renal basement membranes. This stage lasts several months to a year. Once the diuretic phase of acute renal failure begins, the patient may begin to loose massive amounts of fluids through urination. Thus, the problems encountered in this phase may be opposite to those encountered in the oliguric phase of ARF. The patient may actually begin to experience dehydration if the amount of fluid being replaced is not able to meet the pace of urinary excretion. Hypokalemia may be present at this stage as massive amounts of potassium begin to be excreted along with urine. The risks for infection, GI bleeding, skin breakdown, altered nutrition, and sleep disturbances remain problematic.

The recovery phase can last from several weeks to a year. The amount of recovery in the Glomerular filtration rate depends on the number of residual functional nephrons present. If too few nephrons remain, they too eventually begin to sclerosis from the burden of hyperfiltration, and begin to die. This continuing cycle is called the Hyperfiltration theory of Acute Renal Failure, explaining why Chronic Kidney Disease can follow an apparent recovery from ARF.

Dialysis in ARF

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Indications for and timing of initiation of RRT (renal replacement therapy) are also important and somewhat controversial subjects. Widely accepted indications for initiation of RRT include the following:

Volume overload. Any patient who is oliguric < 500 cc of urine in a 24 hour period is at risk for fluid overload and hemodynamic instability

Hyperkalemia (K^+ >6.5 or rising)

Acid-base imbalance

Symptomatic uremia (pericarditis, encephalopathy, bleeding dyscrasia, nausea, vomiting, pruritus)

Uremia (BUN>100). If the creatinine clearance is <30 mg/dL then profound uremia is likely to occur in conjunction with hyperkalemia and acidosis.

Dialyzable intoxications. Drug overdoses need to be evaluated for dialysis due to potential for further nephrotoxic injury from continued exposure to the overdosed drug.

Severe dysnatremia (<115 or >165), and dysthermia may also be appropriate indications for RRT. Significant intoxications with a dialyzable agent (eg, methanol, ethylene glycol, theophylline, aspirin, lithium) may be the strongest indication for emergent dialysis because other effective therapeutic interventions are available for most of other complications of ARF. Volume overload can be treated with nitrates and phlebotomy; hyperkalemia can be treated with calcium, insulin, glucose, bicarbonate, binding resins, and beta agonists (Agraharkar. 2007).

The principal methods of renal replacement therapy (RRT) are intermittent hemodialysis (IHD), continuous venovenous hemofiltration (CVVH), and peritoneal dialysis (PD). Each has advantages and limitations.

IHD is widely available, has only moderate technical difficulty, and is the most efficient way of removing a volume or solute from the vascular compartment quickly. Unfortunately, dialysis-associated hypotension may adversely affect remaining renal function, particularly in patients who are critically ill. This is one reason CVVH is widely recommended in this setting.

Continuous RRT techniques are more expensive and not universally available; however, in addition to avoiding hypotension, they are believed to achieve better control of uremia and clearance of solute from the extravascular compartment. Because it continues around the clock, CVVH is able to remove larger fluid

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volumes, which is a significant advantage with critical care patients on parenteral nutrition and multiple infusions. CVVH may also preserve cerebral perfusion pressure more effectively. A theoretical though contested advantage of CVVH is the clearance of mediators of the inflammatory cascade. Although several studies have sought to directly compare CVVH to IHD, no study has shown a convincing advantage for one therapy over the other; in spite of this, many authorities assert that the choice of IHD over CVVH in the setting of shock would be inappropriate and unethical.

Peritoneal dialysis is inexpensive, widely available, and does not result in hypotension. However, it is not capable of removing large volumes of fluid or solute. Its use may be most common in children.

Slow renal replacement therapies are usually completed in the acute care setting when a patient is not able to tolerate more aggressive dialysis therapy. There are several variations of slow continuous therapies, and most share the same attributes of removing water and solutes over a prolonged period. Most of the therapies require no complex machinery to perform. There are generally four types of slow continuous therapies, continuous arteriovenous hemofiltration (CAVH), continuous venovenous hemofiltration (CVVH), Continuous arteriovenous hemodialysis (CAVHD), and continuous venovenous hemodialysis (CVVHD). A fifth therapeutic modality is called slow continuous Ultrafiltration (SCUF), and removes only fluid and very minimal solutes.

Traditional dialysis tends to cause rapid shifts in fluid and electrolyte balance causing hypotension and other intradialytic complications. Slow continuous therapies are ideal for hemodynamically unstable patients because they do not cause rapid shifts in fluid volumes or electrolytes. The correction of acid base and electrolyte balance happens as the changes are evolving, which produces a more steady state situation. The procedure is highly effective at the correction of pulmonary edema and other hypervolemic states, requiring no special equipment. Finally, the procedure is simple to do. Slow continuous therapies are appropriate for most types of people with many different comorbid conditions, so the potential for utilization of these therapies is enormous.

Slow continuous hemodialysis therapies involve using a dialyzer that is connected to the patient via a vascular access. Vascular access is usually achieved by using a dual lumen catheter inserted into the femoral vein. Arterial access is associated with a higher complication rate. The subclavian and jugular veins can also be used as access sites. The tip of the femoral catheter should optimally lie in the inferior vena cava. The access must be able to deliver an adequate blood flow rate to prevent clotting in the dialyzer circuit. For treatment in which blood flow is not assisted with a blood pump device, an arterial access must be achieved and the patient must have an adequate mean arterial pressure to propel blood through the dialysis circuit.

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Prior to the initiation of the therapy, the blood circuit is primed with heparinized saline. Sterile precautions must be adhered to so that infection is prevented. When performing slow continuous therapies without the aid of a blood pump, the dialyzer must be positioned below the level of the heart to maximize the effects of hydrostatic pressure. The lines and dialyzed should be secured close to the access, but within visualization of staff at all times. Line connections should be checked to make sure that they remain secure throughout the procedure to minimize the risk of blood loss to the patient

A slow infusion of dialysate is circulated through the dialysate compartment of the dialyzer. This allows for greater solute removal than in CAVH or CVVH and is used for patients who are hyperkalemic or acidotic. The composition of the dialysate can be tailored to achieve desired serum concentrations of various electrolytes. In the venovenous form of slow continuous hemodialysis a blood pump is necessary to propel the blood through the dialysis circuit. With continuous arteriovenous hemodialysis, a pump is not used, as the patient's own blood propels the blood through the dialysis circuit. This is due to the higher vascular pressure from the arterial blood compartment.

With slow continuous hemofiltration, a pump may be used if the patient's primary means of access is the venous blood compartment. Arterial access uses the patient's own blood pressure to propel the blood through the blood circuit. Fluid losses can be up to 15 liters per day. With this large amount of fluid loss a certain amount of obligatory solute removal occurs due to solute drag. Higher clearances are achieved with the pump method however and this method is the more favored of the two. This was the first of the slow continuous renal replacement therapies.

Solute clearance may be enhanced by several means. When using CAVH, suction can be applied to the ultrafiltrate port to enhance clearance. Suction will cause an increase in the solute drag across the dialyzer membrane. Solute clearance is also affected by when replacement fluids are administered. Fluids administered pre-dialysis filter tend to achieve higher solute clearances because the viscosity of the blood is greatly diminished, increasing the efficiency at which the blood moves through the filter. The process of adding a pump to the dialysis circuit also improves the efficiency of the treatment and increases solute removal. Finally, another option to increase solute clearance is to increase the dialyzer flow rate.

Slow continuous Ultrafiltration is achieved by either an arterial access or venous access to the patient's blood. All other slow continuous therapies require some type of fluid replacement to be given to the patient. The average maximal fluid removal is 2000ml per hour. Very minimal waste is removed with SCU. SCU is generally used in patients who are hemodynamically unstable or are hypotensive in who electrolyte balance is not a paramount issue. This type of therapy is particularly suited to individuals with congestive heart failure. The excess fluid

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removal can dynamically improve oxygenation and cardiac output in patients with fluid overload.

Slow continuous hemodialysis removes a greater volume of urea and solutes than hemofiltration. This is due to the infusion of dialysate and the resulting diffusion gradient. Hemofiltration does involve some amount of solute removal, but 10-15 liters of fluid would have to be pulled from the patient per day with hemofiltration to achieve effective solute removal. On the average, urea clearances of 18-20ml per minute can be achieved with slow continuous hemodialysis. Urea clearances with hemofiltration average being approximately 50% less. Fluid filtration rates for hemofiltration are about 600ml per hour while hemodialysis rates are 300ml per hour.

One of the main complications of renal replacement therapy is fluid volume deficit. One of the requirements of this procedure is that meticulous intake and output recordings be kept and errors in this process can lead to intravascular hypovolemia if not detected. A potentially common complication of renal replacement therapy is clotting of the blood circuit. When blood is exposed to a foreign substance, the natural tendency is for clotting to occur. Anticoagulation is frequently used to prevent clotting, however it does occur despite interventions to prevent it and some patients are not able to receive anticoagulation further complicating the problems. Issues with blood loss also occur as a result of clotting or can occur if bloodlines accidentally become disconnected. Several different anticoagulation medications and delivery methods may be used in slow renal replacement therapies. Heparin is the most commonly used anticoagulant, but calcium citrate, prostacyclin, and nafomostat mesilate can be used as well. Anticoagulation with citrated preparations requires special dialysate and serum calcium levels must be closely monitored as citrate binds with the body's calcium and can cause hypocalcemia. Anticoagulation methods can be systemic or regional; regional anticoagulation involves the administration of heparin into the blood prior to it entering the dialyzer, and infusion of protamine before the blood is returned to the patient. The treatment may also be conducted heparin free, but the circuit must be rinsed with saline hourly to prevent clotting.

Finally, the potential for hypothermia does exist because blood is being circulated outside the body and large amounts of fluids are being utilized as replacement and dialysate in the procedure.

Conclusion

The prompt recognition of ARF is essential to restoring renal function. ARF carries a high mortality rate, which can be diminished by being alert to early signs of problems in the acute renal failure patient. Nurses can be of particular importance in the health care team by being able to identify and alert providers to complications of ARF. Hopefully, as education increases and medical technology improves patient mortality will decline.

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