Neonatal Acute Kidney Injury

Yenidoğan Akut Böbrek Hasarı

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ABSTRACT

Acute kidney injury (AKI) is characterized by a sudden deterioration in kidney function that results in the accumulation of nitrogenous waste products (e.g., urea) and alters the regulation of extracellular fluid volume, electrolytes, and acid-base homeostasis. Although the criteria for neonatal acute kidney injury have varied, a frequently used definition is a serum creatinine level of more than 1.5 mg/dL.The causes of neonatal acute kidney injury are multiple and can be divided into prerenal, renal, and postrenal categories. Prerenal azotemia is the most common type of acute kidney injury in the neonate and may account for up to 85% of all cases. There are currently no specific medical therapies to treat AKI. The basic approach in management of AKI should be planned according to the underlying etiology. To maximize the chance for survival, the clinician must support the cardiorespiratory system, maintain maximal nutrition, balance homeostasis, and manage the consequences of AKI. The prognosis for neonates with acute kidney injury is variable, and largely related to the infant's underlying medical condition, with mortality rates ranging from 14% to 73%. *(JAREM 2013; 3: 53-9)*

Key Words: Neonatal, acute, kidney injury

ÖZET

Akut böbrek hasarı; böbrek fonksiyonlarında ani bozulma ile karakterizedir. Sonuç olarak nitrojen yıkım ürünlerinin birikimi, ekstrasellüler sıvı hacmi, elektrolit ve asid-baz dengesinin regülasyonunda değişiklikler olur. Neonatal akut böbrek hasarı kriterleri değişmekle beraber, sıklıkla serum kreatinin düzeyinin 1,5 mg/dL'yi geçmesi tanım olarak kabul edilebilir. Neonatal akut böbrek hasarının birçok nedeni vardır ve prerenal, renal ve postrenal olarak kategorilere ayrılabilir. Prerenal azotemi yenidoğan döneminde en sık görülen kategoridir ve tüm vakaların yaklaşık %85'i bu gruptandır. Akut böbrek hasarının halen günümüzde spesifik bir medikal tedavisi yoktur. Hastayı yönetirken temel yaklaşım altta yatan etiyolojiye göre planlanmalıdır. Hastanın yaşama şansını yükseltmek adına klinisyen, bebeğin kardiyopulmoner sistemini desteklemeli, maksimal beslenmeyi sağlamalı, homeostazı dengelemeli ve akut böbrek hasarının sonuçlarını yönetebilmelidir. Akut böbrek hasarı ile başvuran yenidoğanlarda prognoz değişkendir ve çoğunlukla bebeğin altta yatan medikal durumuyla ilişkilidir. Mortalite oranları %14 ile %73 arasında değişmektedir. *(JAREM 2013; 3: 53-9)*

Anahtar Sözcükler: Yenidoğan, akut, böbrek hasarı

DEFINITION

Acute kidney injury (AKI) is characterized by a sudden deterioration in kidney function that results in the accumulation of nitrogenous waste products (e.g., urea) and alters the regulation of extracellular fluid volume, electrolytes, and acid-base homeostasis. Although the criteria for neonatal acute kidney injury have varied, a frequently used definition is a serum creatinine level of more than 1.5 mg/dL (1). Oliguric acute kidney injury is characterized by a urine flow rate of less than 1 mL/kg per hour, whereas in nonoliguric acute kidney injury the urine flow rate is maintained above this level.

In the adult and pediatric populations, classification definitions of AKI are based on SCr (Serum Creatinine) and urine output. The two most common classification systems for severity of AKI are the Risk, Injury, Failure, Loss, and End-Stage Renal Disease (RIFLE) (Table 1) (2) and the Acute Kidney Injury Network (AKIN) (Table 2) (3) classifications. In children, Akcan-Arikan et al. (4) suggested a modified pediatric RIFLE (pRIFLE) classification with a lower cutoff of SCr to achieve the failure (F) category, thereby reflecting the fact that children have a lower baseline SCr. Similar classification definitions of AKI are greatly needed to better describe the incidence and outcomes of AKI in different populations of critically ill neonates.

Despite these working classification systems, the diagnosis of AKI is problematic, because current diagnosis relies on two functional abnormalities: functional changes in SCr [marker of Glomerular Filtration Rate (GFR)] and oliguria. Studies report more than 50% of AKI cases to be nonoliguric, which highlights the insensitivity of oliguria in predicting AKI in neonates.

Both of these measures are late consequences of injury and not markers of the injury itself. The ideal marker to detect AKI should be upregulated shortly after an injury and be independent of the GFR (1). Current studies of urinary and serum biomarkers of AKI promise to improve our ability to diagnose AKI early in its disease process. For example, urine and serum neutrophil gelatinase-associated lipocalin, urine interleukin-18, kidney injury marker 1, and others have been shown to predict which neonates undergoing cardiopulmonary bypass will develop a rise in SCr level greater than 0.5 mg/dL (5, 6). Creating AKI definitions using early injury biomarkers, which can ultimately predict morbidity and mortality, is of paramount importance.

RISK FACTORS AND ETIOLOGY

Risk factors for development of neonatal acute kidney injury include very low birth weight (less than 1500 g), low 5-minute APGAR score, maternal drug administration (nonsteroidal anti-inflammatory drugs and antibiotics), intubation at birth, respiratory

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Table 1. Pediatric-modified RIFLE (p RIFLE) criteria

	Estimated CCl	Urine Output
Risk	eCCl decrease by 25%	< 0.5 mL/kg/h for 8 h
Injury	eCCl decrease by 50%	< 0.5 mL/kg/h for 16 h
Failure	eCCl decrease by %75 or eCCl < 35 mL/min/1.73 m^2	< 0.3 mL/kg/h for 24 h or anuric for 12 h
Loss	Persistent failure > 4 weeks	
End stage	End-stage renal disease (persistent failure > 3 months)	

eCCl: estimated creatinine clearance, p RIFLE: pediatric risk, injury, failure, loss and end-stage renal disease

Table 2. Acute Kidney Injury Network (AKIN) classification/staging system

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of more than or equal to 0.3 mg/dL or increase to more than or equal to 150% to 200% (1.5 to 2-fold) from baseline	Less than 0.5 mL/kg per hour for more than 6 hours
2	Increase in serum creatinine to more than 200% to 300% (>2 to 3-fold) from baseline	Less than 0.5 mL/kg per hour for more than 12 hours
3	Increase in serum creatinine to more than 300% (>3-fold) from baseline (or serum creatinine of more than or equal to 4.0 mg/dL with an acute inrease of at least 0.5 mg/dL	Less than 0.3 mL/kg per hour for 24 hours or anuria for 12 hours

distress syndrome, patent ductus arteriosus, phototherapy, and neonatal medication administration (nonsteroidal anti-inflammatory drugs, antibiotics, diuretics, etc.) (7).

The causes of neonatal acute kidney injury are multiple and can be divided into prerenal, renal, and postrenal categories (Table 3).

PRERENAL AZOTEMIA

Prerenal azotemia is the most common type of acute kidney injury in the neonate and may account for up to 85% of all cases. Prerenal azotemia is characterized by inadequate renal perfusion, which, if promptly treated, is followed by improvements in renal function and urine output.

INTRINSIC (RENAL) ACUTE KIDNEY INJURY

ATN (Acute Tubular Necrosis) is the most common cause of intrinsic acute kidney injury in neonates. The causes of ATN include perinatal asphyxia, sepsis, cardiac surgery, a prolonged prerenal state, and nephrotoxic drug administration. The pathophysiology of ATN is complex and appears to involve renal tubular cellular injury, alterations in adhesion molecules, and changes in renal hemodynamics.

OBSTRUCTIVE (POSTRENAL) ACUTE KIDNEY INJURY

Obstructive acute kidney injury is caused by bilateral urinary tract obstruction and can usually be reversed by relief of the obstruction.

EVALUATION

The pregnancy history, findings on prenatal tests, vital signs, changes in neonatal body weight, physical examination, interventions, and medications prescribed provide important clues about the cause of neonatal AKI. The signs of acute kidney injury may include oliguria, systemic hypertension, cardiac arrhythmia, evidence of fluid overload or dehydration, decreased activity, seizure, vomiting, and anorexia. Laboratory evidence may include elevated serum creatinine and blood urea nitrogen, hyperkalemia, metabolic acidosis, hypocalcemia, hyperphosphatemia, and a prolonged half-life for medications excreted by the kidney (e.g., aminoglycosides, vancomycin).

Serum laboratory tests to be monitored in the infant with AKI include serum sodium, potassium, chloride, bicarbonate, calcium, phosphorus, magnesium, urea, creatinine, uric acid, glucose, blood gases, hemoglobin, and platelets. If urine is available, then urinalysis, urine culture, and a spot urine sample for sodium, creatinine, and osmolality can help for differential diagnosis.

One of the major goals in the initial evaluation of neonatal AKI is to determine whether the kidney is hypoperfused. Several laboratory, clinical, and therapeutic interventions can help to delineate prerenal azotemia from intrinsic AKI (Table 4). These laboratory studies have important limitations in premature infants. Unfortunately, laboratory tests to determine whether elevated SCr is from prerenal azotemia versus intrinsic AKI are insensitive and nonspecific in premature infants because of underdeveloped tubular function. Normal fractional sodium excretion in preterm infants born before 32 weeks' gestation is usually higher than 3% (8). In addition, because of the developmentally regulated limitation of their concentrating capacity and the effects of low protein intake and urea excretion on urine osmolality, the urine-to-plasma creatinine ratio rather than the urine-to-plasma osmolality ratio should be used in newborns to evaluate their renal tubular reabsorptive capacity (9).

A second major goal of AKI evaluation is to detect anatomic causes of AKI, if present. A renal and bladder ultrasound examination should be performed without delay if an obstructive process is suspected and to detect congenital renal abnormalities if present. If hematuria, hypertension, or both are present, the possibility of renal vascular disease should also be considered. Doppler ultrasound examination of renal vessels can be performed if renal vascular thrombosis is suspected.

Table 3. Causes of Acute Kidney Injury in the Newborn*

Prerenal Azotemia	Intrinsic Acute Kidney Injury	Obstructive Renal Failure
Loss of effective blood volume Absolute loss Hemorrhage Dehydration Relative loss ↑ Capillary leak Sepsis NEC RDS ECMO Hypoalbuminemia Renal hypoperfusion Congestive heart failure Pharmacologic agents Indomethacin Tolazoline ACE inhibitors	Acute tubular necrosis Severe renal ischemia Nephrotoxins Infections Congenital infections Pyelonephritis Bacterial endocarditis Renal vascular causes Renal artery thrombosis Renal vein thrombosis DIC Nephrotoxins Aminoglycosides Indomethacin Amphotericin B Radiocontrast dyes Acyclovir Intrarenal obstruction Uric acid nephropathy Myoglobinuria Hemoglobinuria Congenital malformations Bilateral renal agenesis Renal dysplasia Polycystic kidneys	Congenital malformations Imperforate prepuce Urethral stricture PUV Urethral diverticulum Ureterocele Megaureter UPJ obstruction Extrinsic compression Sacrococcygeal teratoma Hematocolpos Intrinsic obstruction Renal calculi Fungus balls Neurogenic bladder

*Textbook of Avery's Disease of the Newborn Ninth Edition, Part XVII, pg 1208

ACE: Angiotensin-converting enzyme, DIC: disseminated intravascular coagulation, ECMO: extracorporeal membrane oxygenation, NEC: necrotizing enterocolitis, PUV: posterior urethral valve, UPJ: ureteropelvic junction

Table 4. Prerenal Azotemia versus Intrinsic Acute Kidney Injury in the Newborn*

	Prerenal Azotemia	Intrinsic Acute Kidney Injury
Urine flow rate (mL/kg/h)	Variable	Variable
Urine osmolality (mOsm/L)	>400	≤400
Urine-to-plasma osmolal ratio	>1.3	≤1.0
Urine-to-plasma creatinine ratio	29.2±1.6	9.7±3.6
Urine [Na^+] (mEq/L)	10-50	30-90
FENa[†] (%)	<0.3 (0.9±0.6)	>3.0 (4.3±2.2)
Renal failure index[‡]	<3.0 (1.3±0.8)	>3.0 (11.6±9.5)
Response to fluid challenge	Improved tachycardia, increased urine output (UOP)	No effect on tachycardia or UOP

†Fractional excretion of sodium (FENa) = (Urine [Na^+]/Serum [Na^+])/(Urine [Cr]/Serum [Cr]) × 100.

‡Renal failure index (RFI) = Urine [Na^+]/(Urine [Cr]/Serum [Cr]).

*Textbook of Avery's Disease of the Newborn Ninth Edition, Part XVII, pg 1210

A voiding cystourethrogram (VCUG) may need to be performed to rule out obstructive uropathy and/or vesicoureteral reflux in some newborns with renal anomalies detected on prenatal ultrasound, especially those with antenatal hydronephrosis.

Radionuclide scintigraphy can be used to demonstrate renal structure and function. Renal function and blood flow can be assessed using isotopes such as DTPA or MAG 3 that are handled by glomerular filtration. The renal cortex can be evaluated using isotopes such as technetium-99m-dimercaptosuccinic acid (DMSA) that bind to renal tubules.

Although these studies are difficult to accomplish in sick infants, they are essential in newborns with prolonged anuria to evaluate ischemic renal damage (cortical necrosis) or urinary tract obstruction with significant hydronephrosis.

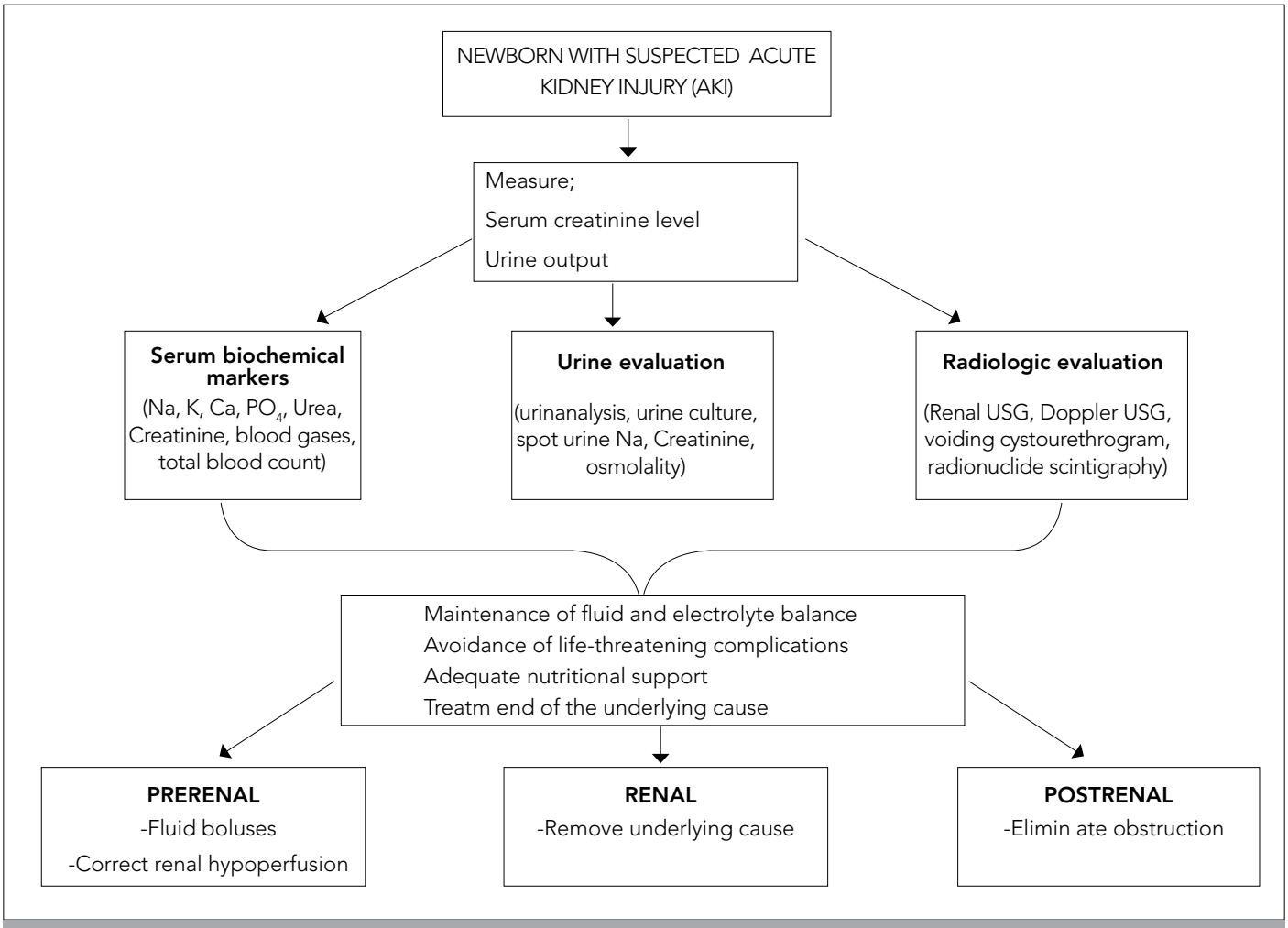

Figure 1. Simple approach for a newborn with suspected AKI

The diagram in Figure 1 shows a simple approach to a newborn with suspicion for AKI.

MANAGEMENT

Medical Management

There are currently no specific medical therapies to treat AKI. The basic approach in management of AKI should be planned according to underlying etiology.

The approach using fluid boluses for prerenal azotemia also serves as the initial management of this condition. If obstruction of the urinary outflow is discovered, then interventions to eliminate the obstruction should be undertaken followed by plans for surgical correction. Polyuria with electrolyte losses can occur after relief of the obstruction; therefore close monitoring of serum electrolytes, especially bicarbonate, and appropriate replacement of these losses are necessary. Besides these specific management options, there are currently no specific medical therapies to treat AKI. To maximize the chance for survival, the clinician must support the cardiorespiratory system, maintain maximal nutrition, balance homeostasis, and manage the consequences of AKI. Dialysis can provide renal suppport to achieve goal-oriented therapies.

Dopamine can increase renal perfusion in the sick preterm and term infant with prerenal azotemia caused by hypoxemia, acidosis, or indomethacin administration (10-12). Although low dose dopamine increases renal perfusion, randomized controlled studies in adults with AKI have reached the same conclusion (13-16). Compared with a placebo, low-dose dopamine does not improve survival, shorten hospital stay, or limit dialysis use. These studies have not been performed in children or neonates.

Diuretics are commonly used for induction of diuresis in critically ill neonates; however, no studies in neonates, children, or adults have shown that diuretics are effective in preventing AKI or improving outcomes once AKI occurs (13). If loop diuretics are to be used in neonates, continuous doses of furosemide may be superior to larger intermittent doses. The authors conclude that those with continuous dosing may have less risk for nephrotoxicity or autotoxicity than occurs with large intermittent doses of this drug (17). The potential toxicity of long-term and aggressive furosemide therapy-including ototoxicity, interstitial nephritis, osteopenia, nephrocalcinosis, hypotension, and persistence of patent ductus arteriosus-should be considered, especially in the preterm newborn (18).

If systemic hypotension develops despite adequate volume administration, early initiation of blood pressure support often establishes appropriate renal perfusion (11, 19). In cases of pres-

Table 5. Medical Management of Hyperkalemia in the Newborn (22)

Drug	Dose	Onset of Action	Duration of Action
Calcium gluconate (10%)	0.5-1 mL/kg (IV over 10 min)	1-5 min	15-60 min
Sodium bicarbonate (3.75% solution)	1-2 mEq/kg (IV over 10 min)	5-10 min	2-6 h
Insulin	1 IU/5 g glucose (IV bolus or continuous infusion)	15-30 min	4-6 h
Glucose	≤14 mg/kg/min (IV bolus or continuous infusion)	15-30 min	4-6 h
Furosemide	1 mg/kg dose or as continuous infusion	5-10 min	2-3 h
Sodium polystyrene sulfonate	1 g/kg dose every 6 h as needed (orally/rectally)	1-2 h	4-6 h

sor-inotrop–resistant hypotension and shock, a brief course of low-dose hydrocortisone has been demonstrated to be effective in restoring systemic perfusion and renal function in preterm neonates (20). Other management goals include maintaining blood oxygen content, providing blood products for specific indices, limiting severe acidosis, and maintaining normal serum albuminemia (at least 2.0 mg/dL, but preferably 2.5 mg/dL).

Hypertension is common in neonates with AKI. It can be caused by increased renin release in malformed or damaged kidneys or may be secondary to increased intravascular volume from a lack of free water clearance. If hypertension is due to fluid overload, inducing free water clearance with diuretics or fluid removal with dialysis will address its cause. Calcium-channel blockers work by selectively causing vasodilatation of the venous system. Short-acting calcium-channel blockers are reliable, have a quick onset of response, and are well tolerated. Long-acting calcium-channel blockers (e.g., amlodipine) take longer to take effect, but they provide less lability with longer dosing intervals. β-Blockers (propranolol or labetolol) are also commonly used to treat hypertension in neonates. Use of ACE-I (angiotensin converting enzyme inhibitor) in children with ischemic AKI should be avoided, because it can produce further renal hypoperfusion and can alter intrarenal hemodynamics in an already injured kidney.

Managing fluids in the critically ill neonate with AKI can be difficult. These infants may require large volumes of fluid to maintain adequate nutrition and hematologic indices and to provide appropriate medications. However, these fluids can be detrimental in a child with oliguria or anuria, because they can cause congestive heart failure and pulmonary failure. Therefore, once adequate intravascular volume has been restored, the aim should be to prevent severe fluid overload (by limiting crystalloid infusions) and maximize nutritional supplements concentration.

Management of electrolyte disorders can usually be achieved by attention to electrolyte intake during the initial course of AKI with frequent evaluation and specific therapies. Most cases of hyponatremia are due to water overload and less commonly due to low total body sodium content. Attention to fluid status is critical to determine the cause and proper therapy of hyponatremia. In cases of nonsymptomatic hypervolemic hyponatremia (serum sodium concentrations usually between 120 and 130 mEq/L), restriction of free water intake is recommended. If hyponatremia at this level results in clinical signs and symptoms (e.g., lethargy, seizures) or serum sodium concentration falls to less than 120 mEq/L, use of 3% sodium chloride should be considered.

Severe hyperkalemia is a life-threatening medical emergency. Hyperkalemia which is unresponsive to medical management is one of the most common indications for peritoneal and hemodialysis in the newborn (18, 21). Measures to remove potassium from the body include oral or rectal sodium polystyrene (Kayexalate), loop diuretics to enhance potassium excretion (if not anuric), and dialysis. Several methods to move potassium from the extracellular to the intracellular compartment are available, including albuterol inhalation, sodium bicarbonate, and insulin plus glucose. Adequate ionized calcium levels for cardioprotection should be sought in the context of hyperkalemia (Table 5) (22).

Hyperphosphatemia is common in AKI and should be treated with low phosphorus intake. Breastmilk contains low phosphorous and low potassium compared with other neonatal infant formulas. For this reason, feeding with breastmilk is recommended for newborns with AKI. Significant elevations in serum phosphate represent a risk of development of extraskeletal calcifications of the heart, blood vessels, and kidneys in the newborn, especially when the calcium-phosphorus product exceeds 70 (23). Calcium carbonate may be used as a phosphate-binding agent in infants whose phosphorous intake exceeds excretion. Although rarely an indication for dialysis without fluid overload or hyperkalemia, severe hyperphosphatemia is best treated with dialysis.

The incidence of hypocalcemia is low in neonates with severe and prolonged AKI, especially in those who develop an inability to convert 25-hydroxy–vitamin D to 1,25-hydroxy–vitamin D. Ionized calcium should be measured when low total calcium levels and concomitant hypoalbuminemia are encountered, because the latter can affect total calcium levels. If ionized calcium is decreased and the newborn is symptomatic, 100 to 200 mg/kg of calcium gluconate should be infused over 10 to 20 minutes and repeated every 4 to 8 hours as necessary. If hypocalcemia is severe, oral or intravenous calcitriol can be administered to increase intestinal reabsorption of calcium.

Normal acid–base homeostasis depends on the kidney's ability to reabsorb bicarbonate; therefore infants with AKI commonly have a non–anion gap metabolic acidosis. Replacement with bicarbonate or acetate as a base is indicated in infants with AKI to avoid or treat metabolic acidosis. In infants with severe respiratory failure, large doses of bicarbonate should be avoided because they can result in increased carbondioxide retention. Metabolic acidosis should be treated aggressively in infants with severe pulmonary hypertension, because an acidic environment can worsen this condition.

Nutritional goals in infants with AKI are similar to those of infants without AKI. Commonly parenteral nutrition, feeds, or both will

need to be concentrated to avoid excessive fluid gains. If nutritional goals cannot be achieved because of oliguria or ongoing fluid overload, the potential risks of dialysis therapy versus the potential risks associated with inadequate calorie and protein administration should be discussed with the parents. If a neonate is receiving continuous peritoneal dialysis or hemodialysis, an additional 1 g/kg/day of protein is needed to supplement the protein losses that occur with these forms of dialysis (24, 25).

In a neonate with AKI, careful assessment of medication dosing is important. Because many drugs are excreted in the urine, impaired metabolism or clearance from the kidneys can cause drug accumulation and adverse side effects. In infants receiving dialysis, pharmacokinetic properties of drugs (e.g., volume of distribution, protein binding, size, charge), dialysis modality (peritoneal dialysis versus hemodialysis) and interval of dialysis (intermittent versus continuous) will affect drug availability (26).

RENAL REPLACEMENT THERAPY

Renal replacement therapy should be considered if maximum medical management fails to maintain acceptable fluid and electrolyte levels. The two purposes of renal replacement therapy are ultrafiltration (removal of water) and dialysis (removal of solutes). The indications for the initiation of renal replacement therapy include hyperkalemia, hyponatremia with symptomatic volume overload, acidosis, hypocalcemia, hyperphosphatemia, uremic symptoms, and an inability to provide adequate nutrition due to the need for fluid restriction in the face of oliguria.

Peritoneal dialysis is the most commonly used renal replacement modality in the neonatal population because it is technically easier and does not require vascular access or anticoagulation. For this procedure, the hyperosmolar dialysate is repeatedly infused into and drained out of the peritoneal cavity through a surgically placed catheter, accomplishing ultrafiltration and dialysis. Cycle length, dwell volume, and the osmolar concentration of the dialysate can be varied to accomplish the goals of therapy. The relative contraindications to peritoneal dialysis include recent abdominal surgery, necrotizing enterocolitis, pleuroperitoneal leakage, and ventriculoperitoneal shunting.

Continuous renal replacement therapy (CRRT) is becoming a more frequently used therapeutic modality in the neonate whose condition is unstable (27). For this procedure, the patient's blood is continuously circulated through a pump-driven extracorporeal circuit containing a highly permeable hemofilter. In continuous venovenous hemofiltration (CVVH), an ultrafiltrate of plasma is removed, a portion of which is returned to the patient in the form of a physiologic replacement fluid. In continuous venovenous hemodialysis (CVVH-D), countercurrent dialysate is used rather than replacement fluid to achieve solute removal. The major advantage of CRRT is the ability to carefully control fluid removal, which makes this modality especially useful in the neonate with hemodynamic instability. The main disadvantages are the need to achieve and maintain central vascular access and the need for continuous anticoagulation. Intermittent hemodialysis is a less commonly used but technically feasible mode of renal replacement therapy in the neonatal population. Hemodialysis involves intermittent 3- to 4-hour treatment periods in which fluids and solutes are rapidly removed from the infant by using an extracorporeal dialyzer, with clearance achieved by the use of countercurrent dialysate. The chief advantage of hemodialysis is the ability to rapidly remove solutes and fluids, a characteristic that makes this modality the therapy of choice in neonatal hyperammonemia (28). The main disadvantages are the requirement for central vascular access and the hemodynamic instability and osmolar shifts that may occur with rapid solute and fluid shifts. The ability to provide renal replacement therapy may be limited by the ability to place and maintain intravascular or peritoneal dialysis access in the very small premature neonate. If dialysis access cannot be established, care of the infant with acute kidney injury is limited to maximal supportive medical management with meticulous attention to fluid and electrolyte balance.

PROGNOSIS

The prognosis for neonates with acute kidney injury is variable, and largely related to the infant's underlying medical condition, with mortality rates ranging from 14% to 73% (29). Bolat et al found that newborns with very low birth weight, bronchopulmonary dysplasia, antenatal steroid, high creatinine level, blood urea nitrogen and potassium, low serum sodium level, anuria, dialysis and mechanical ventilation and hypotension requiring inotropic support were significantly associated with the mortality of the infants (30). In general, infants with prerenal acute kidney injury who receive prompt treatment for renal hypoperfusion have an excellent prognosis. Infants with postrenal acute kidney injury related to congenital urinary tract obstruction have a variable outcome which depends on the degree of associated renal dysplasia. Infants with intrinsic acute kidney injury have significant risks of morbidity and mortality. A study of 23 infants who received peritoneal dialysis during the first month of life showed that at 1 year 30% were on dialysis, 9% had chronic renal failure, 26% had made a full renal recovery, and 35% had died in the neonatal period (31). There was a substantial difference in outcome according to the underlying cause of acute kidney injury; neonates with renal structural anomalies had a 17% mortality rate, and those with ATN had a 55% mortality rate (31). Prominent risk factors for progressive kidney disease included proteinuria (urine protein/Cr ratio greater than 0.6 at 1 year of age), serum Cr greater than 0.6 mg/dL at 1 year of age, and body mass index greater than the 85th percentile. Other long-term sequelae seen in survivors of neonatal acute kidney injury include hypertension, an impaired capacity for urinary concentration, renal tubular acidosis, and impaired renal growth.

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Yazarlar herhangi bir çıkar çatışması bildirmemişlerdir.

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